Review Article

Coronary Artery Disease: Comprehensive Evaluation by Cardiovascular Magnetic Resonance Imaging

RS Tan,[1] *MBBS, MRCP*, KKW Chen,[2] *MBChB, MRCP, FRCR*

Abstract

Cardiac magnetic resonance (CMR) imaging has emerged as an important non-invasive cardiac imaging modality. A versatile technique, it has the potential for comprehensive evaluation of coronary artery disease (CAD): cardiac morphology and function, myocardial perfusion, myocardial viability, coronary artery visualisation and atherosclerotic plaque characterisation. Some of these techniques – ventricular function and myocardial viability assessment – have already made mainstream clinical impact. Other techniques have yielded promising initial results, and will become increasingly accepted with technical refinement in scanner hardware and software. This article reviews the current status of clinical CMR imaging for diagnosis of CAD.

Ann Acad Med Singapore 2004;33:437-43

Key words: Coronary artery, Perfusion, Ventricular function, Viability

Introduction

Coronary artery disease (CAD) is a major health problem worldwide. With advances in CAD intervention and improvements in treatment and management of CAD, there has been a dramatic increase in the survival rate of patients. This, combined with the effects of an ageing population, has resulted in a growing number of patients with chronic ischaemic heart disease. Detection of disease and evaluation of its severity and complications are important to clinicians. Cardiovascular magnetic resonance (CMR) imaging has the potential for comprehensive non-invasive evaluation of CAD: cardiac morphology and function, myocardial perfusion, myocardial viability, coronary artery visualisation and atherosclerotic plaque characterisation. Comparison with other imaging modalities is contentious and frequently reflects individual institutional expertise. The comparison is inevitably ever in a state of flux as technical improvements and experience evolve in each modality (Table 1).

Modern CMR scanners, enhanced by fast magnetic gradient performance, innovative pulse sequence design and parallel imaging techniques, routinely produce high quality images within reasonable examination times. Unlike invasive cardiac catheterisation, computed tomography (CT) and radionuclide imaging, there is no exposure to ionising radiation or nephrotoxic contrast media. Patient-friendly scanner design, use of silent CMR pulse sequences and the availability of non-ferromagnetic physiologic monitors and resuscitation equipment enhance patient comfort and safety. Due to the potential for device malfunction and induction of ectopy or heat injury, CMR should not be performed in patients with implanted cardioverter-defibrillators, active permanent pacemakers or ventricular assist devices. The presence of well-deployed (and well-epithelised) coronary artery stents[1] and mechanical prosthetic heart valves are not contraindications to CMR.

Ventricular Morphology and Function

Most clinical CMR imaging protocols include ECG-gated cine images acquired in planes parallel and orthogonal to the left ventricular major axis, similar to standard echocardiographic views (Fig. 1). Abnormalities of left ventricular wall motion and thickening can be identified by studying endocardial and epicardial contour excursion. The presence of myocardial thinning suggests prior infarction. Complications of myocardial infarct such as mural thrombus, aneurysmal dilatation, ventricular septal and papillary muscle rupture are readily recognised (Fig. 2). Grid tagging (using spatial modulation of magnetisation pulse sequences) improves assessment of wall motion abnormality.

[1] Department of Cardiology
National Heart Centre, Singapore
[2] Department of Diagnostic Radiology
Singapore General Hospital, Singapore
Address for Reprints: Dr Tan Ru San, Department of Cardiology, National Heart Centre, Mistri Wing, 17 Third Hospital Avenue, Singapore 168752.
Email: Tan_Ru_San@nhc.com.sg

Table 1. Imaging Modalities in the Assessment of Coronary Artery Disease

	CMR	Echo/MCE	Nucl/PET	CT	Cath
Morphology	++++	++++	+	++++	++
Function	++++	+++	+++	+	+++
Myocardial strain	++*	+++	–	–	–
Haemodynamics	++	++++	–	–	++++
Perfusion	+++	+++	++++	+	+++†
Viability	++++	+++	++++	+	–
Coronary artery	++	+	–	+++	++++
Plaque imaging	++	+	–	++	++++‡
Molecular imaging	++§	++	++	–	–

– Not applicable; + Limited role; ++ Fair; +++ Good; ++++ Excellent
* Myocardial tagging
† Doppler flow wire
‡ Intravascular ultrasound
§ Spectroscopy
Cath: cardiac catheterisation; CMR: cardiac magnetic resonance; CT: multi-slice computed tomography; Echo/MCE: echocardiography and microbubble contrast echocardiography; Nucl/PET: radionuclide perfusion imaging, radionuclide ventriculography and positron emission tomography

CMR can image the heart with large fields of view, free of acoustic window limitations. It provides precise and highly reproducible quantitative measurements of chamber volumes and function.[2] Typically, a stack of short axis cine images encompassing both ventricles from base to apex is acquired in about 10 breath-holds, each lasting about 10 seconds. Ventricular volumes are measured using Simpson's method of discs (Fig. 3). This method makes no geometric assumption, and is the gold standard for assessment of the right ventricle, the complex shape of which defies mathematical modelling. In patients who cannot maintain adequate apnoea, real-time image acquisition may be used, with minor loss of spatial resolution that does not affect volume assessment significantly.[3]

Interstudy reproducibility of left ventricular volumes and ejection fraction is excellent.[4,5] Serial changes in individual patients' cardiac volumes and function, due to natural disease progression or therapeutic intervention, can be followed longitudinally with great reliability. Similarly, the calculated sample size requirements for comparative studies of ventricular function and mass are considerably smaller with CMR versus echocardiography. This translates into lower study costs.[5]

Myocardial Ischaemia

The ischaemic cascade describes the temporal sequence of events that occur during myocardial ischaemia: coronary flow heterogeneity (subendocardial hypoperfusion followed by transmural hypoperfusion), diastolic dysfunction (abnormal myocardial relaxation), systolic dysfunction (diminished contraction), ECG changes and angina.[6] CMR detection of differential myocardial perfusion and contractile abnormalities with pharmacological stress (it is impractical to exercise within the CMR scanner) are 2 strategies for diagnosing myocardial ischaemia.

Vasodilator Stress Myocardial Perfusion Imaging

In myocardial perfusion imaging, temporally resolved multi-slice images of the heart are acquired during first pass following intravenous administration of paramagnetic contrast agent gadolinium-diethylenetriaminepentaacetate (Gd-DTPA). The latter increases T1-weighted tissue signal intensity (SI) as it transits the heart (Fig. 4). With qualitative assessment, reversible perfusion defects (areas of decreased SI) visualised during maximal vasodilatory pharmacological stress (induced with intravenous adenosine) but not during rest indicate ischaemia. The superior spatial resolution afforded by CMR allows delineation of perfusion differences between subendocardial and subepicardial myocardium. Hypoperfused myocardium characteristically appears as a persistent dark subendocardial rim. Experience is, however, needed to discriminate dark subendocardial ring image artifacts, which are typically transient, from true regions of ischaemia.

CMR perfusion studies may be analysed using a semi-quantitative approach. Regions of interest can be drawn within any area of myocardium, and SI versus time curves generated. Semi-quantitative indices of myocardial perfusion include peak SI, first derivative of upslope (derived from curve fit of first-pass SI data), time to peak, mean transit time and area under SI curve. Of these, the regional myocardial upslope parameter, normalised to the upslope parameter of left ventricular blood pool, has been most widely investigated. Decreased regional myocardial perfusion reserve index, i.e. the ratio of stress upslope parameter to its rest value, has been demonstrated to correlate with epicardial artery stenosis.[7-9]

Gd-DTPA is an extravascular contrast agent. Besides myocardial blood flow, vascular permeability and contrast medium distribution also influence SI. Due to the non-linear relationship between SI and myocardial blood flow, quantitation of absolute myocardial blood flow requires complex mathematical analyses.[10] It is anticipated that in the near future, automated analysis software programmes will become commercially available for routine clinical quantitative myocardial perfusion analysis.

High-dose Dobutamine Stress CMR

Dobutamine has been used for stress wall motion imaging in conjunction with cine CMR. Nagel et al[11] compared high-dose dobutamine stress echocardiography (with harmonic imaging) and CMR in 208 consecutive patients with suspected CAD who all underwent subsequent coronary angiography. CMR yielded better image quality with improved sensitivity (86% vs 74%) and specificity (86% vs

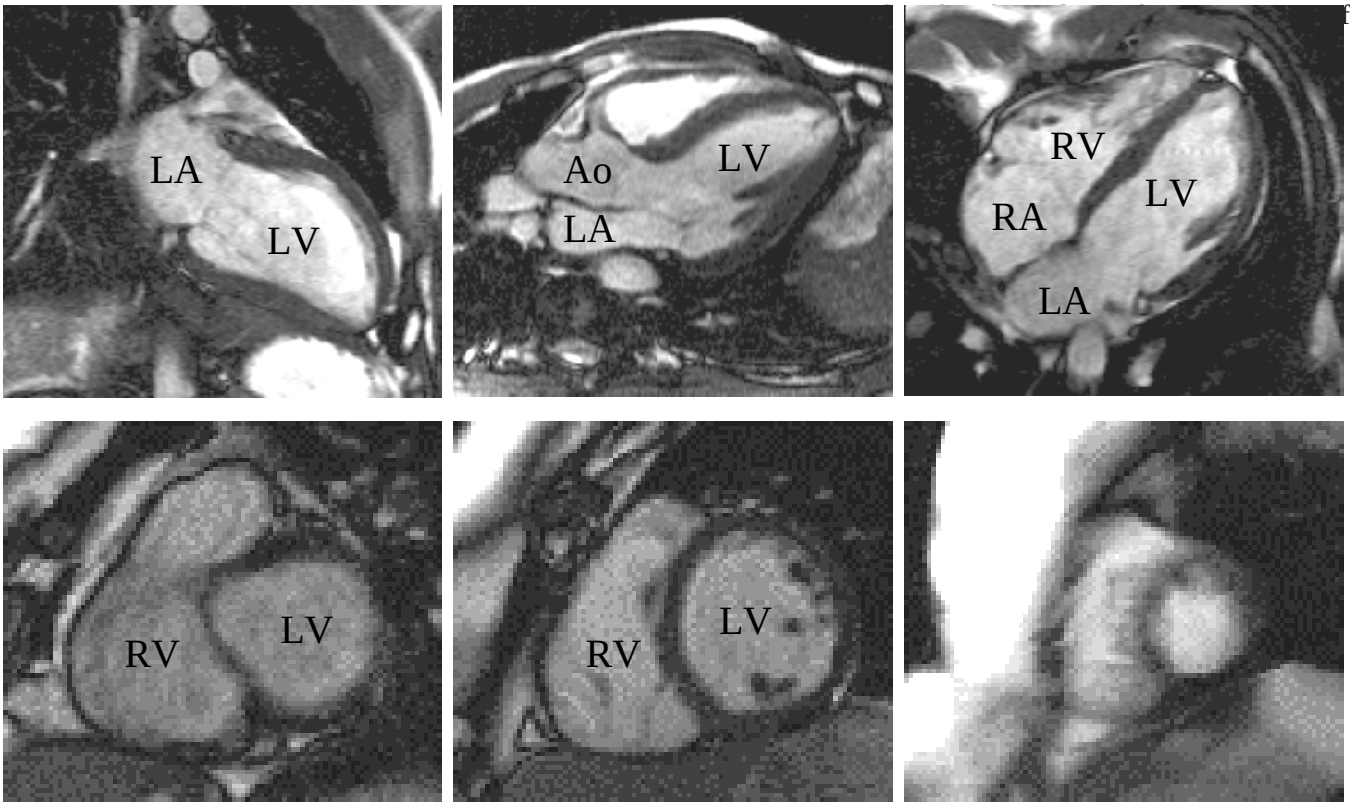

Fig. 1. Typical CMR cardiac imaging planes. Long-axis views: 2-chamber, 3-chamber (left ventricular outflow tract view) and 4-chamber views (top row, left to right). Short-axis views: basal, mid and apical views (bottom row, left to right). Ao: Aorta; LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.

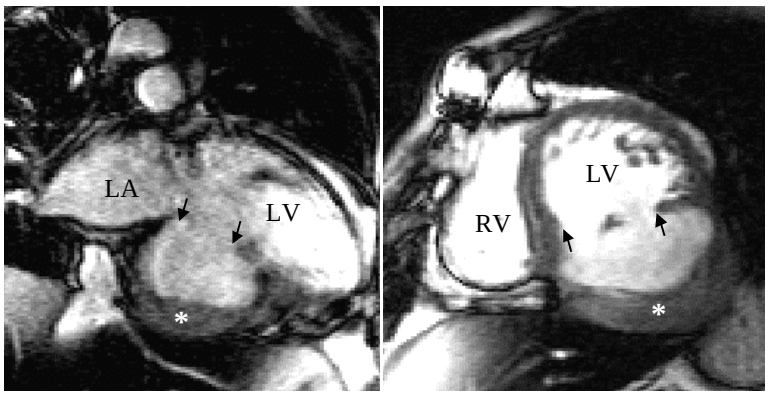

Fig. 2. Chronic inferior myocardial infarct with pseudoaneurysm formation. Two-chamber (left) and short-axis (right) views. The pseudoaneurysm neck (arrows) is narrow relative to the body. Within its cavity, note mural thrombus (asterisk).

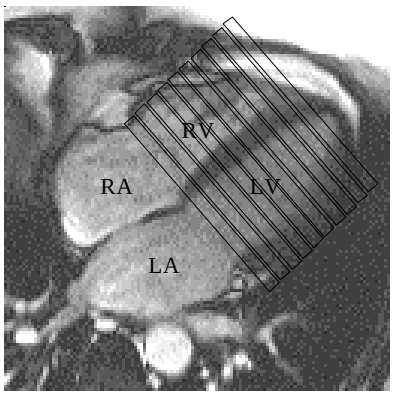

Fig. 3. Simpson's method of discs. Four-chamber cine image of the heart, from which a stack of short-axis slices, each with equal slice thickness and interslice gap, is positioned to cover the left and right ventricles from the atrioventricular junction to the apex. At each slice, endocardial contours are traced at end-diastole and end-systole. The ventricular volume contained within each slice is the product of the planimetered area and the interslice distance between contiguous images. End-diastolic and end-systolic ventricular volumes are derived from summation of the slice volumes at the appropriate cardiac phases; and ejection fraction calculated accordingly.

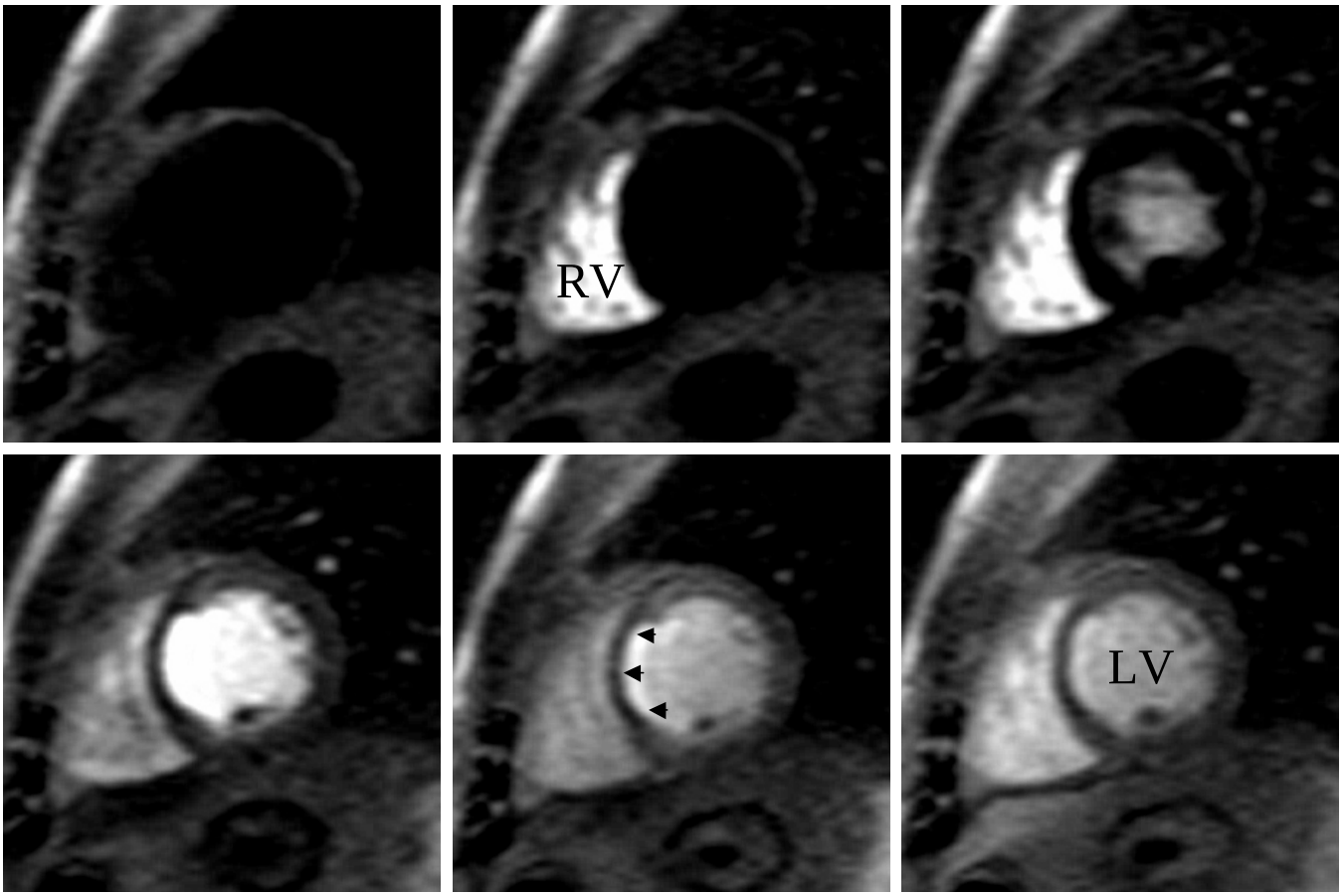

Fig. 4. Stress myocardial perfusion imaging demonstrating septal wall ischaemia. Mid-ventricular short-axis images acquired sequentially during first pass infusion of Gd-DTPA, at the 1st, 7th, 10th (top row, left to right), 25th, 30th and 45th (bottom row, left to right) heart beats after start of scan. Note passage of contrast from the right ventricle to the left ventricle. Normal myocardium is brighter than hypoperfused myocardium, and attains the peak intensity faster. The persistent dark rim at the septal subendocardium (arrowheads) represents ischaemia. Typically, CMR perfusion imaging is performed during maximal vasodilatory stress (intravenous adenosine 140 μg/kg/min for 6 minutes; image acquisition at 3 minutes) and rest. Two separate boluses of Gd-DTPA are given at a sufficient time interval apart to allow for washout of the initial dose. Subsecond rapid CMR images of Gd-DTPA first pass through the myocardium are acquired in multiple slices (usually 3 to 4 short axis slices) at a temporal frequency of once every 1 to 2 heart beats, for about a minute.

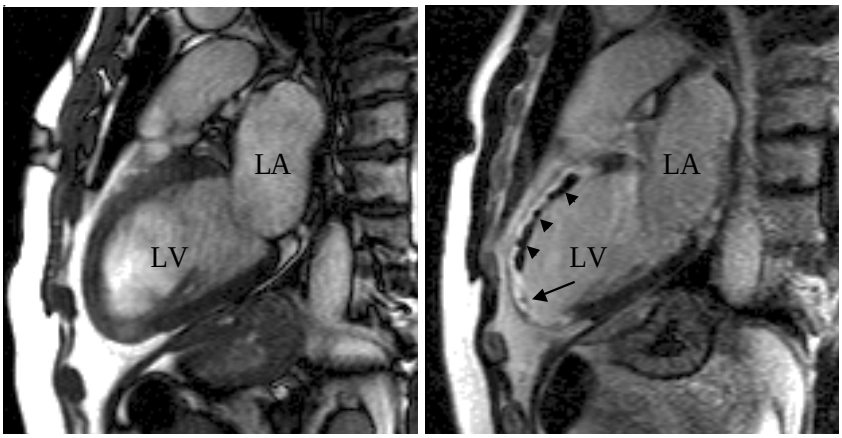

Fig. 5. Recent anterior myocardial infarct. Two-chamber cine CMR image (left), and delayed hyperenhancement image acquired using a special pulse sequence to null myocardial signal intensity (right). Note transmural hyperenhancement of anterior and apical walls (arrow) indicating infarct. The dark area within the infarct core (arrowheads) indicates microvascular obstruction.

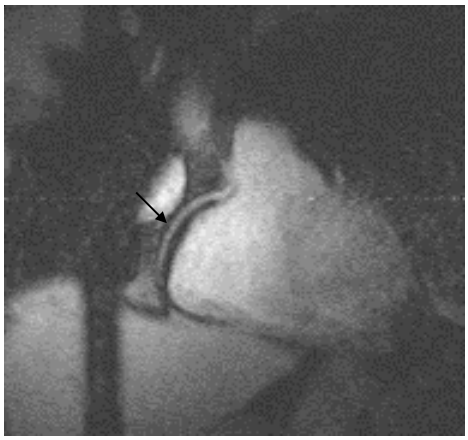

Fig. 6. Respiratory-gated free-breathing CMR imaging of the right coronary artery (arrow) in a healthy subject. The dark vertical band at the right hemidiaphragm represents the navigator echo used for respiratory gating.

patients with uninterpretable echocardiographic images, Hundley et al[12] established that inducible ischaemia on dobutamine stress CMR was predictive of major adverse cardiac events.

Improved endocardial visualisation is the main CMR advantage in these studies. The validity of this conclusion needs to be re-examined given the widespread use of recently Food Drug Administration (FDA)-approved microbubble echocardiographic contrast agents, which opacify the left ventricular cavity and greatly enhance echocardiographic endocardial border definition.

Myocardial Viability

With advances in medical management, there has been a gradual decline in deaths from myocardial infarction, and a striking increase in the incidence of heart failure among survivors. Detection of dysfunctional but viable myocardium is important because of the potential for restoration of contractile function following revascularisation. Identification of non-viable myocardium is equally important: patients are prevented from undergoing high-risk revascularisation procedures that are unlikely to confer survival benefit.

Thinned myocardium represents chronic healed transmural infarction. On cine CMR imaging, a wall segment with end-diastolic wall thickness ≤6 mm connotes low likelihood of functional improvement with revascularisation.[13,14] This criterion is, however, neither sensitive (recent transmural infarct with incomplete remodelling may not appear thinned) nor specific (viability may be maintained in thinned and akinetic segments in some cases) for myocardial viability. Additionally, low-dose dobutamine stress (up to 10 μg/kg/min) CMR may be used to assess contractile reserve, a marker of myocardial viability.

Delayed Hyperenhancement

The best way to detect non-viable myocardium with CMR is with a technique called delayed hyperenhancement (DE), whereby heart imaging is carried out 10 to 30 minutes after intravenous Gd-DTPA administration. DE exploits the altered magnetisation properties induced by increased tissue concentrations of Gd-DTPA in acute and chronic infarct tissues. In acute myocardial infarct, there is a relatively large volume of distribution and avid uptake of Gd-DTPA into the oedematous tissue. In chronic myocardial infarct with fibrous scarring, there is increased extracellular water surrounding the collagenous fibres, which augments Gd-DTPA retention within the scar tissue. By either means, great signal contrast between normal myocardium and infarct tissue is created as more Gd-DTPA washes out of normal myocardium and accumulates in infarcted areas (Fig. 5). In animal studies, the extent of DE correlates very well with myocardial necrosis on histological examination.[15,16] In humans, the transmural extent of DE, in the setting of either chronic or acute myocardial infarction, is inversely related to regional and global contractile improvement after revascularisation.[17,18] Transmural extent of DE less than 25% best predicts post-revascularisation contractile recovery. DE compares favourably with positron emission tomography (PET), the acknowledged gold standard for the assessment of viability.[19]

In acute or recent myocardial infarct, imaging may in some cases reveal areas of markedly reduced SI within the bright infarcted areas. These represent regions of microvascular obstruction ("no reflow") within the core of the infarct (Fig. 5). These areas are not perfused by the contrast medium, and will enhance extremely slowly. With time, these areas necrose and are resorbed as the myocardium undergoes remodelling. The presence of microvascular obstruction is an independent predictor of adverse prognosis.[20]

Coronary Artery Anatomy

Many CMR techniques are used to image the coronary arteries. The plethora of methods underscores the daunting challenges facing the imager: small coronary vessel size, vessel tortuousity, cardiac and respiratory motion. Ultrafast pulse sequences are obligatory. Images are ECG-gated and acquired during diastole, where there is least cardiac motion. Breath-holding is used to eliminate the effects of respiratory motion. The breath-hold, typically about 30 seconds, imposes a constraint on vessel image resolution and the volume of coverage. Contrast medium may be utilised: image acquisition during first pass of Gd-DTPA increases vessel signal-to-noise ratio, and may plausibly improve coronary artery delineation. Alternatively, respiratory gating using a "navigator" pulse sequence which tracks diaphragmatic motion to ensure image acquisition only during end-expiratory apnoea, allows imaging with free-breathing. This enhances spatial resolution and vessel coverage, but at the expense of increased scan time (Fig. 6).

Clinical Results

Multiple single-centre studies of CMR coronary artery imaging using various techniques have produced promising results, with sensitivities approaching 90% for diagnosis of significant proximal coronary artery stenosis.[21-25] Coronary artery stenosis appears as a discrete area of signal attenuation or dropout distal to the obstruction. Grading of stenosis severity is, however, less satisfactory as flow artifacts secondary to vessel tortuousity also produce signal dropout, resulting in overestimation of severity. In the only multi-centre trial of CMR coronary artery imaging, Kim et al[25] demonstrated the reproducibility of a standardised respiratory-gated clinical imaging protocol in multiple

centres in different countries. In this study, proximal, but not distal, segments of the coronary arteries were reliably visualised. Up to 16% of vessel segments were uninterpretable due to poor image quality. Although sensitivity of 93% was seen for identifying significant CAD, specificity was disappointingly low at 42%.

More technical refinements are required before CMR coronary artery assessment can become routine. Current niche indications for CMR include evaluation of anomalous coronary arteries[26] (for which the 3-dimensional nature of CMR is a clear advantage over conventional angiography) and possibly coronary bypass graft patency.[27]

Coronary Blood Flow

CMR measurement of coronary blood flow provides important physiological information, especially in coronary stenoses of intermediate severity. Good correlation between coronary flow reserve (ratio of coronary blood flow at maximal vasodilatory stress to that at rest) measured by magnetic resonance phase shift velocity mapping and invasive Doppler flow wire has been established in normal volunteers and patients with CAD. Among patients who have previously undergone percutaneous coronary intervention, CMR-assessed coronary flow reserve ≤ 2 has been reported to be sensitive and fairly specific for detecting flow-limiting restenotic lesions.[28]

Atherosclerotic Plaque

High resolution, multi-contrast CMR imaging can non-invasively image vulnerable atherosclerotic plaques and characterise plaques in terms of their various components (lipids, fibrous cap, thrombus, calcium). Animal and ex vivo studies have shown remarkable correlation between CMR plaque appearance and histology. Most clinical studies have been validated on non-coronary vessels such as the iliac, abdominal aortic and carotid vessels.[29,30] To date, there have been few studies on coronary vessel wall imaging in normal human volunteers and anecdotal reports on diseased clinical patients.[31]

Imaging of extracoronary vessel wall, e.g. the carotid artery, provides excellent surrogate evidence of coronary atherosclerotic burden. Plaque extent and volume can be accurately measured, and its stability assessed.[29] This valuable information offers a unique opportunity for studying disease pathophysiology, and may be used to follow natural disease progression and therapeutic intervention.[32]

Conclusion

CMR offers comprehensive evaluation of ischaemic heart disease. Quantitative CMR assessment of ventricular function is the gold standard. DE has developed, in a few short years, into a highly accurate and impressive technique for myocardial viability assessment. CMR myocardial perfusion imaging is very promising and is at an advanced stage of development. It will become more widely accepted as quantitative analysis software becomes available. At present, non-invasive coronary artery and atherosclerotic plaque imaging have limited niche applications. To translate the encouraging initial results of specialised laboratories into the wider clinical arena will require more technical refinement.

CMR is a hybrid subspecialty that benefits from the collaboration between cardiologists and radiologists, whose expertise and experience are brought together. CMR's unique capabilities complement, rather than compete, with other imaging modalities. The biggest clinical challenge facing CMR today is to integrate itself into clinical diagnostic and management pathways. With continual advances in the CMR machine performance, use of higher magnetic field strengths and novel image processing techniques, this will become a clinical reality in the near future.

REFERENCES

1. Hug J, Nagel E, Bornstedt A, Schnackenburg B, Oswald H, Fleck E. Coronary arterial stents: safety and artifacts during MR imaging. Radiology 2000;216:781-7.
2. Dulce MC, Mostbeck GH, Friese KK, Caputo GR, Higgins CB. Quantitation of the left ventricular volumes and function with cine MRI: comparison of geometric volumes and three-dimensional data. Radiology 1993;188:371-6.
3. Yang PC, Kerr AB, Liu AC, Liang DH, Hardy C, Meyer CH, et al. New real-time interactive cardiac magnetic resonance imaging system complements echocardiography. J Am Coll Cardiol 1998;32:2049-56.
4. Grothues F, Smith GC, Moon JC, Bellenger NG, Collins P, Klein HU, et al. Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects in patients with heart failure or left ventricular hypertrophy. Am J Cardiol 2002;90:29-34.
5. 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; 2:271-8.
6. Nesto RW, Kowalchuk GJ. The ischemic cascade: temporal sequence of hemodynamic, electrocardiographic and symptomatic expressions of ischemia. Am J Cardiol 1987;59:23-30.
7. Al-Saadi N, Nagel E, Gross M, Bornstedt A, Schnackenburg B, Klein C, et al. Noninvasive detection of myocardial ischemia from perfusion reserve based on cardiovascular magnetic resonance. Circulation 2000;101:1379-83.
8. Al-Saadi N, Nagel E, Gross M, Schnackenburg B, Paetsch I, Klein C, et al. Improvement of myocardial perfusion reserve early after coronary intervention: assessment with cardiovascular magnetic resonance imaging. J Am Coll Cardiol 2000;36:1557-64.
9. Schwitter J, Nanz D, Kneifel S, Bertschinger K, Buchi M, Knusel PR, et al. Assessment of myocardial perfusion in coronary artery disease by magnetic resonance: a comparison with positron emission tomography and coronary angiography. Circulation 2001;103:2230-5.

10. Jerosch-Herold M, Seethamraju RT, Swingen CM, Wilke NM, Stillman AE. Analysis of myocardial perfusion MRI. J Magn Reson Imaging 2004;19:758-70.
11. Nagel E, Lehmkuhl HB, Bocksch W, Klein C, Vogel U, Frantz E, et al. Noninvasive diagnosis of ischemia-induced wall motion abnormalities with use of high-dose dobutamine stress CMRI: comparison with dobutamine stress echocardiography. Circulation 1999;99:763-70.
12. Hundley WG, Hamilton CA, Thomas MS, Herrington DM, Salido TB, Kitzman DW, et al. 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;100:1697-702.
13. Klow NE, Smith HJ, Gullestad L, Seem E, Endresen K. Outcome of bypass surgery in patients with chronic ischaemic left ventricular dysfunction: predictive value of MR imaging. Acta Radiol 1997;38: 76-82.
14. Cwajg JM, Cwajg E, Nagueh SF, He ZX, Qureshi U, Olmos LI, et al. End-diastolic wall thickness as a predictor of recovery of function in myocardial hibernation: relation to rest-redistribution Tl-201 tomography and dobutamine stress echocardiography. J Am Coll Cardiol 2000;35: 1152-61.
15. Kim RJ, Fieno DS, Parrish TB, Harris K, Chen EL, Simonetti O, et al. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. Circulation 1999;100:1992-2002.
16. 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;36:1985-91.
17. 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;343:1445-53.
18. Choi K, Kim RJ, Gubernikoff G, Vargas JD, Parker MS, Judd RM. The transmural extent of acute myocardial infarction predicts long term improvement in contractile function. Circulation 2001;104:1101-7.
19. Klein C, Nekolla SG, Bengel FM, Momose M, Sammer A, Haas F, et al. Assessment of myocardial viability with contrast-enhanced magnetic resonance imaging: comparison with positron emission tomography. Circulation 2002;105:162-7.
20. Wu KC, Zerhouni EA, Judd RM, Lugo-Olivieri CH, Barouch LA, Schulman SP, et al. Prognostic significance of microvascular obstruction by magnetic resonance imaging in patients with acute myocardial infarction. Circulation 1998;97:765-72.
21. Sandstede JJ, Pabst T, Beer M, Geis N, Kenn W, Neubauer S, et al. Three-dimensional MR coronary angiography using the navigator technique compared with conventional coronary angiography. AJR Am J Roentgenol 1999;172:135-9.
22. van Geuns RJ, de Bruin HG, Rensing BJ, Wielopolski PA, Hulshoff MD, van Ooijen PM, et al. Magnetic resonance imaging of the coronary arteries: clinical results from three-dimensional evaluation of a respiratory gated technique. Heart 1999;82:515-9.
23. Regenfus M, Ropers D, Achenbach S, Kessler W, Laub G, Daniel WG, et al. Noninvasive detection of coronary artery stenosis using contrast-enhanced three-dimensional breath-hold magnetic resonance coronary angiography. J Am Coll Cardiol 2000;36:44-50.
24. van Geuns RJ, Wielopolski PA, de Bruin HG, Rensing BJ, Hulshoff M, van Ooijen PM, et al. MR coronary angiography with breath-hold targeted volumes: preliminary clinical results. Radiology 2002;217: 270-7.
25. Kim WY, Danias PG, Stuber M, Flamm SD, Plein S, Nagel E, et al. Coronary magnetic resonance angiography for the detection of coronary stenosis. N Engl J Med 2001;345:1863-9.
26. Bunce NH, Lorenz CH, Keegan J, Lesser J, Reyes EM, Firmin DN, et al. Coronary artery anomalies: assessment with free-breathing three-dimensional coronary MR angiography. Radiology 2003;227:201-8.
27. Wintersperger BJ, Engelmann MG, von Smekal A, Knez A, Penzkofer HV, Hofling B, et al. Patency of coronary bypass grafts: assessment with breath-hold contrast-enhanced MR angiography – value of a non-electrocardiographically triggered technique. Radiology 1998;208: 345-51.
28. Hundley WG, Hillis LD, Hamilton CA, Applegate RJ, Herrington DM, Clarke GD, et al. Assessment of coronary artery restenosis with phase-contrast magnetic resonance imaging measurements of coronary flow reserve. Circulation 2000;101:2375-81.
29. Yuan C, Mitsumori LM, Beach KW, Maravilla KM. Carotid atherosclerotic plaque: noninvasive MR characterization and identification of vulnerable lesions. Radiology 2001;221:285-99.
30. Fayad ZA, Nahar T, Fallon JT, Goldman M, Aguinaldo JG, Badimon JJ, et al. In vivo magnetic resonance evaluation of atherosclerotic plaques in the human thoracic aortic: a comparison with transesophageal echocardiography. Circulation 2000;101:2503-9.
31. Fayad ZA, Fuster V, Fallon JT, Jayasundera T, Worthley SG, Helft G, et al. Noninvasive in vivo human coronary artery lumen and wall imaging using black-blood magnetic resonance imaging. Circulation 2000;102:506-10.
32. Corti R, Fuster V, Fayad ZA, Worthley SG, Helft G, Smith D, et al. Lipid lowering by simvastatin induces regression of human atherosclerotic lesions: two years' follow-up by high-resolution noninvasive magnetic resonance imaging. Circulation 2002;106:2884-7.