

Cardiovascular Magnetic Resonance for the Interventional Cardiologist

a report by

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Cardiovascular magnetic resonance (CMR) has found widespread use as an important tool in the cardiologists' armamentarium for several years now, mainly because of superior diagnostic accuracy and ability to perform complete anatomical and functional assessment in a single study without ionising radiation. The following will summarise specific uses of CMR for the interventional cardiologist.

Theoretical and Practical Concepts^{1,2}

Magnetic resonance imaging (MRI) is based on detection of protons (¹H) from water and fat in the body. ¹H in water contain molecular magnets that align with the magnetic field of the MRI scanner. The intrinsic angular momentum of ¹H results in precession (or rotation) around the axis of the scanner's magnetic field, referred to as 'spin'. When a weaker perpendicular field is applied transiently then ¹H spins rotate together, emitting a coherent oscillating signal that decays in amplitude and coherence with time. The decay of amplitude (T1 relaxation) and coherence (T2 relaxation) is unique to each specific tissue and generates radiofrequency (RF) energy. Receiver coils positioned near the region of interest collect RF signal to gather information on tissue translated into an image.

In practical terms, patient preparation begins by confirming absence of major contraindications (cerebrovascular clips, cochlear implants, ocular metallic fragments and most pacemakers/defibrillators). Many coronary and peripheral stents are safe for CMR immediately after implantation, as are several nitinol-based devices, such as septal occluders. Serum creatinine is assessed since nephrotoxicity of gadolinium (Gd)-based contrast is comparable with iodine-based agents at comparable volumes, although much smaller volumes are used in CMR (20–40cc). Patients undergoing CMR stress should withhold heart rate-reducing medications or caffeine-containing substances. Patients have a 20 gauge intravenous catheter positioned in an arm vein, and are fitted with vectorcardiographic chest leads for ECG gating and a flexible phased array cardiac receiver coil on the precordium. Brachial cuff

pressure and digital pulse oximetry are periodically measured throughout. Visual and auditory communication is maintained. Over the 20–45 minute examination (depending on the indication) most imaging sequences are performed during repeated six to 12 second breath holds on expiration.

Cardiac Function

CMR combines excellent contrast with superior spatial and temporal resolution, providing images of exceptional quality throughout the cardiac cycle, enabling accurate volumetric quantification for global and regional functional analysis. Myocardial tagging offers insights into wall strain patterns revealing intrinsic mechanisms of ventricular function. Velocity encoding techniques accurately assess blood flow to measure valvular function and shunts.

Standardised measurement of global and regional left and right ventricular (RV) function is commonly performed in sequential short axis slices of 6–10mm thickness with 0–10mm gaps. In end-systole and end-diastole, endocardial and epicardial borders are traced in each of the 8–12 short axis slices, and the left ventricle (LV) is segmented following the 17 segment model of the American Heart Association. Applying a modified Simpson's rule, left and RV end-systolic and end-diastolic volumes, ejection fraction, and cardiac output are obtained. LV mass is derived from volume. LV regional wall thickening is determined per segment. Volumetric ventricular quantification is more reliable and reproducible than geometric assumptions used in planar imaging techniques (contrast ventriculography, radionuclide scintigraphy, and echocardiography).³ Diastolic function is assessed by velocity encoding CMR and derived from rates of inflow of blood into the LV and RVs. Velocity encoding CMR also provides the basis for pressure gradient and flow measurements pivotal in the evaluation of valvular stenosis and insufficiency, as well as shunt estimation in congenital cardiovascular disease (CVD). The ability to precisely determine cardiovascular anatomy and physiology, and

provide 3D models that can be rotated into all planes are becoming central to interventionalists in the planning of percutaneous valvular procedures.

Myocardial Ischaemia

Flow-limiting coronary artery stenosis is identified as a perfusion deficit or a wall motion abnormality inducible by stress. CMR is particular as it accurately identifies both perfusion and wall motion defects, potentially improving diagnostic accuracy. Pharmacologic stress is induced by increasing heart rate and contractility (dobutamine) or by vasodilatation (adenosine or dipyridamole), with protocols and specifics identical to other stress imaging techniques.

Gadolinium (Gd) contrast first-pass CMR perfusion imaging provides superior spatial (in the order of 2mm²) and temporal resolution for the diagnosis of ischaemic heart disease. Contrast media is injected as an intravenous bolus and circulates to the coronary arteries increasing T1 signal in perfused myocardium. T1 weighted pulse sequences identify perfusion deficits at first-pass during stress (hyperemia) and rest reflecting either hypoperfused viable myocardium or scar tissue. Late enhancement imaging is performed 10 minutes later to differentiate hypoperfused viable myocardium from scar (discussed later).

Analysis is performed according to the AHA 17 segment model either by visual assessment or quantitative measurement of peak signal intensity or single change over time.⁴ CMR first pass perfusion imaging has been repeatedly validated in animal models.⁵ CMR is significantly better than single photon emission computed tomography (SPECT) at identifying greater than 70% diameter stenoses on quantitative coronary angiography (QCA) (areas under the ROC curves of 0.90 for CMR versus 0.73 for SPECT).⁶ CMR yielded a sensitivity of 90% and specificity of 85% for detecting angiographically significant coronary stenoses. When compared with positron emission tomography (PET) imaging, stress perfusion CMR yielded a sensitivity of 91% and a specificity of 94% with an area under the ROC curve of 0.937. In a multicentre study, the clinical usefulness of stress-perfusion CMR has been validated in multicentre studies.⁸ Rest first-pass perfusion CMR is useful for the identification of microvascular dysfunction and obstruction.⁹

Diagnosis of ischaemic heart disease is also performed by CMR by identification of inducible wall motion abnormalities. Segmental wall motion and systolic wall thickening are compared at rest and each successive stage of stress. Dobutamine stress echocardiography (ECG) has been validated as a

useful screening tool for patients with suspected coronary artery disease (CAD), but is limited by 10–15% non-diagnostic studies.¹⁰ Dobutamine stress CMR is superior to echocardiography for sensitivity (86 versus 74%), specificity (86 versus 70%) and accuracy (86 versus 73%).¹¹ Furthermore, CMR yields diagnostic studies in 94% of patients with non-diagnostic stress echocardiography.¹²

Myocardial Necrosis and Viability

In daily practice, DE-CMR is used to differentiate stunned from infarcted myocardium in acute patients, and to predict response to coronary revascularisation in chronic LV systolic dysfunction. While several techniques extrapolate myocardial necrosis from wall thickness or thickening, perfusion or tracer uptake, or improvements in contraction with dobutamine, CMR is unique in precisely identifying presence and absence of living cardiomyocytes. Delayed enhancement CMR (DE-CMR) has been extensively validated in animal and human studies. When compared with the fine details and area of necrosis by triphenyl tetrazolium chloride (TTC) staining, DE-CMR precisely mirrored histological findings in animal models.¹³ While both acute myocardial infarction (AMI) and severe ischaemia manifest loss of systolic function, necrosis identified by DE-CMR consistently differentiates myocardial stunning from infarct.¹³ Discrimination of reversible from irreversible injury is achieved by DE-CMR in both acute and chronic settings.¹⁴ Infarct size determined by DE-CMR is highly reproducible, and correlates closely with cardiac enzyme rise in the setting of AMI.^{15,16} The transmural extent of necrosis identified by DE-CMR is strongly correlated to improvement in global and regional systolic function including recovery from stunning in AMI and success of revascularisation in chronic ischaemic cardiomyopathy.^{17,18} Superior spatial resolution of CMR allows identification of as little as 2g of necrosis, compared with 10g for nuclear techniques.¹⁹ Previously unrecognised microinfarction and subendocardial necrosis are identified, portending important prognostic implications.²⁰

DE-CMR is routinely performed at our institution in patients with AMI or chronic ischaemic cardiomyopathy to predict success of revascularisation and determine best therapeutic strategy. After a 10-minute delay, IV Gd-diethylenetriaminepentaacetic acid (DTPA) distributes to where myocardial cells are absent (scar in chronic infarct) or where their sarcolemmal membranes are disturbed (necrosis in acute infarct). In T1-weighted imaging, inversion time (TI) is set to null normal myocardium and view Gd-DTPA-rich scar or necrosis as a bright signal at least two standard deviations brighter than remote normal myocardium (and typically at least five times

brighter). Adjustment of TI is straightforward and critical in insuring reproducibility of infarct size measurements.¹⁵ Each segment is attributed a transmural score for necrosis (0%; 1–25%; 26–50%; 51–75%; 76–100%). Potential for functional recovery is determined based on transmural necrosis, with >50% transmural necrosis carrying less than 20% potential for improvement, based on the work of Kim et al., referenced above.¹⁸

Atherosclerotic Plaque Burden and Characterisation

CMR provides accurate assessment of vessel lumen, vessel wall, and atherosclerotic plaque areas and volumes compared with histology, with errors within 5% and high inter-observer reproducibility.^{21,22} Arguably more important than atherosclerosis burden is the biology within plaque. Features of plaque causing acute thrombosis include a large lipid-rich

core, thin fibrous cap, and active inflammation.²³ CMR has the ability to characterise tissue within plaque (i.e. lipid, fibrous, calcified tissues, and haemorrhage) as well as the presence of inflammatory cells.^{24,25} Paramagnetic markers of molecular processes within plaque detectable by CMR are a burgeoning area of intense research.²⁶ At present, characterisation of atherosclerosis by CMR is mostly restricted to larger vessels close to the surface of the body. Work is in progress to develop novel receiver antennas able to image atherosclerosis from within using intravascular MRI (IVMRI) to achieve optimal signal to noise ratio and identify vulnerable characteristics of smaller arteries deep within the body.²⁷

Cardiovascular magnetic resonance has become a key component in the daily care of patients suffering from CVD. Interventional cardiologists now have a powerful tool to aid in diagnosis, risk stratification, and optimisation of treatment strategies. ■

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