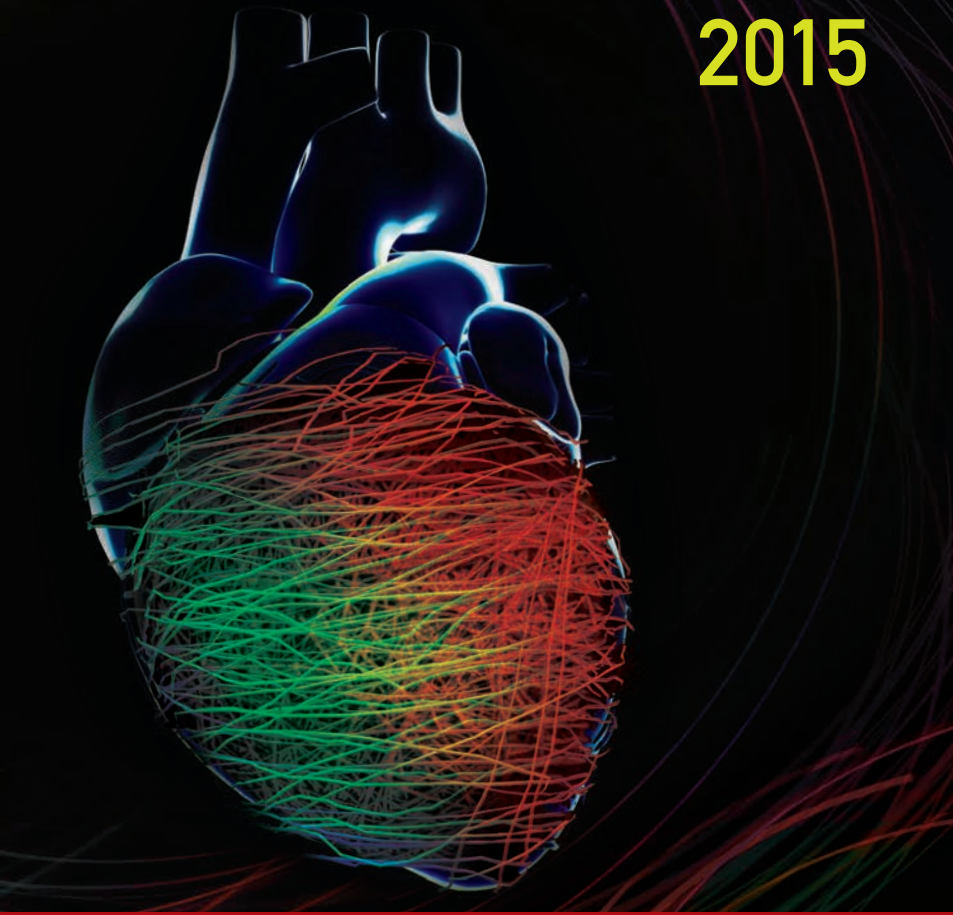
Malaysian Consensus Statement on the

UTILISATION OF CARDIAC MAGNETIC RESONANCE

2015



Ministry of Health Malaysia

National Heart Association of Malaysia

SCIM
Society of Cardiac Imaging Malaysia

COLLEGE OF RADIOLOGY
Academy of Medicine of Malaysia

Committee

WRITING COMMITTEE

Dr. Chua Seng Keong (Chairperson)

Consultant Cardiologist
Park City & Ara Damansara Medical Centre
Kuala Lumpur

Dr. Ainol Shareha Sahar

Consultant Cardiologist
Department of Cardiology
Hospital Pulau Pinang
Pulau Pinang

Dr. Alan Fong Yean Yip

Consultant Cardiologist
Department of Cardiology
Sarawak General Hospital Heart Centre
Sarawak

Dr. Amran Abdul Rahman

Consultant Radiologist
KPJ Ampang Puteri Specialist Hospital
Selangor Darul Ehsan

Assoc. Prof. Dr. Azarisman Shah Mohd Shah

Consultant Cardiologist and Senior Lecturer
Department of Internal Medicine
International Islamic University Malaysia
Pahang

Dr. Effarezan Abdul Rahman

Cardiology Specialist and Senior Lecturer
Cardiology UiTM
Selayang Campus UiTM Medical Faculty
Selangor Darul Ehsan

Dr. Emily Tan Lay Koon

Consultant Cardiologist
Department of Cardiology
National Heart Institute
Kuala Lumpur

Dr. Kevin Louis Joseph

Consultant Cardiologist and General Physician
Kevin Specialist Heart Centre
Pantai Hospital Ipoh
Perak Darul Ridzuan

Dr. Leong Ming Chern

Paediatric Cardiologist
Paediatric and Congenital Heart Centre
National Heart Institute
Kuala Lumpur

Dr. Martin Wong Ngie Liong

Consultant Paediatric Cardiologist
Paediatric Cardiology Unit
Sarawak General Hospital Heart Centre
Sarawak

Dr. Marymol Koshy

Head of Medical Imaging Unit
Faculty of Medicine
Universiti Teknologi MARA
Selangor Darul Ehsan

Dr. Nor Ashikin Md Sari

Cardiologist and Senior Lecturer
Faculty of Medicine
University Malaya
Kuala Lumpur

Dr. Nor Hanim Mohd Amin

Consultant Cardiologist
Department of Cardiology
Sarawak General Hospital Heart Centre
Sarawak

Dr. Norzailin Abu Bakar

Consultant Radiologist and Lecturer
Department of Radiology
Universiti Kebangsaan Malaysia Medical Centre
Kuala Lumpur

Dr. Ravinderjit Singh

Consultant Cardiologist
KPJ Klang Specialist Hospital
Selangor Darul Ehsan

Dr. Shamruz Khan Akerem Khan

Consultant Cardiologist
Subang Jaya & Ara Damansara Medical Centre
Selangor Darul Ehsan

Dr. Suzana Ab. Hamid

Consultant Radiologist and Senior Medical Lecturer
Department of Imaging
Faculty of Medicine & Health Sciences
Universiti Putra Malaysia
Selangor Darul Ehsan

Professor Dr. Yang Faridah Bt Abdul Aziz

Consultant Radiologist
Department of Biomedical Imaging
Faculty of Medicine
University of Malaya
Kuala Lumpur

Dr Yusri Mohammed

Consultant Radiologist
Department of Diagnostic Imaging
Hospital Serdang
Selangor Darul Ehsan

Rationale &
Terms of reference

RATIONALE AND PROCESS OF DEVELOPMENT OF CONSENSUS

Cardiac magnetic resonance (CMR) offers a new dimension in imaging the heart and provides new insights to cardiovascular diseases. In certain aspects it has now become the gold standard in non-invasive imaging. However awareness of CMR in Malaysia remains poor despite being in clinical use for many years. This is additionally confounded by the fact that CMR is an expensive imaging modality with limited expertise in Malaysia.

Recognising this clinical need, the inaugural consensus statement aims to bring about the country's CMR experts and practitioners representing the various health care providers together to create this awareness, to help produce a reference document that will help educate and guide the busy clinician in the utility of CMR.

While formulating this consensus document, ample liberty has been taken to be as inclusive as possible having all the CMR practitioners, cardiologists and radiologists alike to contribute to the best of their abilities, recognising that the CMR training in the country is still varied and heterogenous.

This document is intended to be clinically oriented to further the understanding of CMR, highlighting particular indications and situations where CMR is clinically most helpful. To facilitate ease of reference, the chapters are written in a modular format and meant to be standalone, hence there are some intended repetitions throughout the document emphasising the important aspects of CMR.

There were also rigorous discussions and debates considering the current CMR uptake in the country prior to arriving at the current recommendations for training and competence. This was done clearly recognising that

setting too strict a standard would be counterproductive to the development and growth of CMR discipline. This document is not meant to be authoritative and certainly as the discipline grows with time, the recommended standards will have be revisited again and made in line with international guidelines.

Last but not least I would like to express my heartfelt gratitude to the authors for their unwavering efforts in punctually completing their tasks and chapters.

"Knowledge rests not upon truth alone, but upon error also."

Carl G. Jung

Dr. Chua Seng Keong
Chairperson

TERMS OF REFERENCE

Target population:

Adults with ischaemic and non-ischaemic cardiomyopathies, grown-ups with congenital heart diseases.

Target audience:

Paediatricians, physicians, radiologists, paediatric and adult cardiologists managing cardiovascular diseases.

The consensus statement on the Utilisation of Cardiac Magnetic Resonance 2015 may be obtained from the National Heart Association of Malaysia's website (http://www.malaysianheart.org/) and the Academy of Medicine Malaysia's website (http://www.acadmed.org.my/).

Disclosure:

No conflict of interest declared by any of the committee members.

Source of funding:

This consensus statement on the use of cardiac magnetic resonance was made possible by the National Heart Association of Malaysia (NHAM).

Disclaimer:

The content and recommendations made in this consensus statement are based on currently available scientific data and best clinical practice recommendations from internationally recognised bodies. Its use is for the sole purpose of physicians (cardiologists/radiologists) and radiographers trained to use this modality within Malaysia. Clinical judgement is to prevail in all decisions and should not replace individual responsibility, especially with regards to the safety of users and patients/clients.

Copyright ownership:

Messages

MESSAGE FROM THE DIRECTOR GENERAL OF HEALTH, MALAYSIA

Cardiovascular diseases are undoubtedly the leading cause of death worldwide and certainly in Malaysia. In dealing with the increasing cardiovascular disease burden, it is paramount that contemporary clinical cardiology practice mirrors the ever-growing scientific medical knowledge. Cardiac magnetic resonance (CMR) being the latest addition to the clinical armamentarium of cardiovascular imaging is certainly no different. The importance of CMR is such that today it has already become the gold standard clinical test in some cardiac diseases.

As much as CMR is clinically needed, it has not taken off as expected. Formal institutional services were already available in Sarawak General Hospital since 2003 and 12 years on, we are still challenged by the limited availability of trained physicians, technicians and CMR capable MRI scanners. At the time of writing, there were only just over 20 practicing trained experts scattered in a handful of tertiary or quaternary care institutions throughout Malaysia.

I laud the combined efforts of these CMR specialists representing all the healthcare institutions of the country from the Ministry of Health to university hospitals and private hospitals having taken the challenging effort to come up with this inaugural consensus statement. It is hoped that this consensus statement will be useful beyond just being a reference, generating interest and awareness for this recent imaging modality amongst physicians and patients with cardiac diseases and setting the academic platform for future collaboration with the two stakeholder societies i.e. the National Heart Association of Malaysia and the College of Radiology Malaysia.

In time, it is hoped that these efforts will translate to an increase in allocation of resources leading to the growth of CMR services, thus providing an avenue for the experts to ensure the most updated CMR practices in Malaysia in the years to come.

Lastly, I thank the National Heart Association of Malaysia, its sister society the Society of Cardiac Imaging Malaysia, and the College of Radiology for spearheading this important initiative. This indeed is an exciting and evolving modality to improve cardiovascular healthcare in our country.

Datuk Dr. Noor Hisham Bin Abdullah
MD, MS, AM, FAMM
Director General of Health
Ministry of Health Malaysia

MESSAGE FROM THE PRESIDENT OF THE NATIONAL HEART ASSOCIATION OF MALAYSIA

I am pleased to write a foreword for the first edition of the Consensus Statement on the Utilisation of Cardiac Magnetic Resonance (CMR). As part of the ongoing mission of the National Heart Association of Malaysia (NHAM) to set the standards in cardiovascular care in the country, we are proud to have undertaken this project with the help of the country's experts from the College of Radiology and from NHAM's subsociety the Society of Cardiac Imaging Malaysia.

CMR is the latest non-invasive imaging technology to have emerged in the recent years hence it is only timely for the field of cardiovascular imaging to come to the forefront of cardiology. CMR being technology which is still evolving it would certainly require updates from time to time. Therefore it is truly the aspiration of NHAM for this consensus statement to be the beginning of many more fruitful collaborations to come.

This would be yet another new milestone achieved in terms of collaboration with the College of Radiology Malaysia having collaborated in 2008 and yet again this year on the Consensus Statement on the Utilisation of Cardiac CT. Endeavours such as these are consistent with our society's mission to promote quality cardiovascular care through education, research and to influence health care policies. NHAM will continue to give its fullest commitment to support similar activities in the future to all its enthusiastic members.

Lastly I would like to express my highest acknowledgement of the efforts of the chairperson and all the committee members who have worked selflessly to bring this consensus statement to fruition.

Dr. Rosli Mohd Ali
MD, MRCP, AM, FNHAM, FAPSIC, FAsCC
President,
National Heart Association of Malaysia

MESSAGE FROM THE PRESIDENT OF THE COLLEGE OF RADIOLOGY

It gives me great pleasure to write this foreword accompanying the 1st consensus statement of Cardiac Magnetic Resonance (CMR) in Malaysia.

CMR is – without argument - a single technology that can assess ventricular function, cardiac morphology, the vasculature, perfusion, viability, and metabolism allowing a rather comprehensive cardiac examination.

The major advantage of CMR is the usage of non-ionising radiation and the lack of known biological damage. With sophisticated computer technology and the introduction of ECG-gating, the issue of blurring due to cardiac motion is resolved.

CMR has a distinct advantage over echocardiography and nuclear scintigraphy. There are fewer artifacts and unlimited acquisition windows. As a non-invasive procedure, CMR has positioned itself to become a "one-stop diagnostic modality," offering extensive results during a single examination currently only available through a combination of multiple and separate, diagnostic procedures.

CMR of congenital heart disease is another interesting field that has developed in parallel to the mainstream CMR. Now, CMR is accepted as a useful clinical and research tool in patients with virtually all forms and types of congenital heart disease, ranging in age from newborns to adults. Today, CMR is gradually replacing diagnostic cardiac catheterisation in a variety of clinical circumstances.

In the not so distant future, it is possible that CMR may be used for routine screening of cardiac diseases. It may be possible to know not only ventricular volumes and function but also cardiac valve physiology, pericardial health, complete coronary artery anatomy, myocardial thickness, perfusion, and viability – in other words – anything you want to know about the heart!

Finally, my sincere thanks to the team of radiologists (from College of Radiology) and cardiologists (from National Heart Association of Malaysia) who has worked tirelessly to complete this momentous document.

Congrats everyone!

Dr. Abdul Rahman Mohamad
MBBS, MMed Radiology, AM
President, College of Radiology
Academy of Medicine of Malaysia

TABLE OF CONTENTS

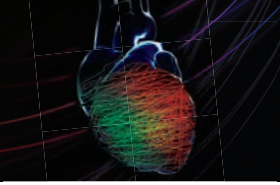
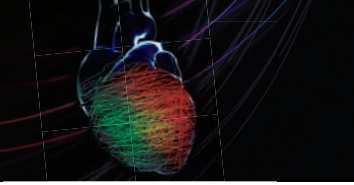


TABLE OF CONTENTS

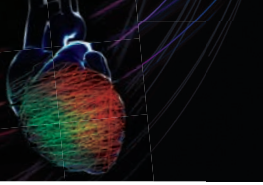
ABBREVIATIONS



ARVC/D	Arrhythmogenic right ventricular cardiomyopathy/dysplasia
ASD	Atrial septal defects
CMR	Cardiac magnetic resonance
CRT	Cardiac resynchronisation therapy
ECG	Electrocardiogram
EGE	Early gadolinium enhancement
ELC	Ethanol-induced cardiomyopathy
Gd	Gadolinium
GFR	Glomerular filtration rate
GRE	Gradient echo
HCM	Hypertrophic cardiomyopathy
ICD	Implantable cardioverter/defibrillator
LGE	Late gadolinium enhancement
LV	Left ventricular
LVOT	Left ventricular outflow tract
MR	Magnetic resonance
MRS	Magnetic resonance spectroscopy
MRA	MR angiography
MRI	Magnetic resonance imaging
MPR	Multiplanar reformation
NSF	Nephrogenic systemic fibrosis
PPCM	Peripartum cardiomyopathy
RF	Radiofrequency
RV	Right ventricular
SCMR	Society for Cardiovascular Magnetic Resonance
SPECT	Single-photon emission computed tomography
SSFP	Steady-state free precession
SCD	Sudden cardiac death

Introduction

INTRODUCTION



Clinical use of cardiac magnetic resonance (CMR) for rudimentary structure and function began in the 1980s and then progressed quickly to the contrasted study for myocardial viability and perfusion in the 1990s.[1] From a 0.1 Tesla MRI in the early days, technology has now progressed such that the use of a 3.0 Tesla scanners and dedicated third party cardiac analysis software are not uncommonly encountered in Malaysia. CMR, amongst all the imaging modalities, is slated to undergo the biggest expansion in the next 10 years.[2]

Its use has evolved to be what can be deemed as a One Stop Shop imaging capable of imaging the whole heart, large vessel structures and masses, being useful in acute and chronic myocardial infarct imaging, coronary artery ischaemia assessment, valvular diseases and cardiomyopathies. Being free of radiation, it is indispensable in the serial monitoring of older children and adults with congenital heart diseases[3] where a combination of flow, left and right ventricular function and morphological assessment serve to guide treatment options.

While CMR can be adequately performed with most scanners of field strengths of 1.5 Tesla, the use of a 3.0 Tesla scanner certainly gives a higher signal to noise ratio at a small cost of increased artifacts. As far as the other hardware requirements are concerned, a body or cardiac coil is mandatory. Though it is too simplistic to assume a minimum required number of 'Radiofrequency (RF) channels', newer centres are recommended to have a scanner with 16 or more RF channels[2] whereas older scanners can at least perform a simple 'structure and function' and viability test. In addition, independent third party dedicated CMR software are available to provide additional features beyond stock standard analysis such as the quantification of scar, and quantifying perfusion defects and myocardial iron content. Furthermore, these software are compatible with Digital Imaging and Communications in Medicine (DICOM) data across different scanners.

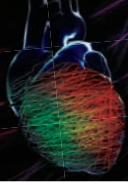
INTRODUCTION

Paul C. Lauterbur and Sir Peter Mansfield were awarded the Nobel Prize in Medicine in 2003 for their discoveries concerning magnetic resonance imaging.

Uses of CMR

1) Morphology study of heart and large vessels
2) Gold standard in left and right ventricular function assessment
3) Coronary ischaemia
   a. Wall motion – Dobutamine stress
   b. Perfusion - Adenosine
4) Cardiomyopathies
5) Valvular diseases
6) Congenital Heart Diseases

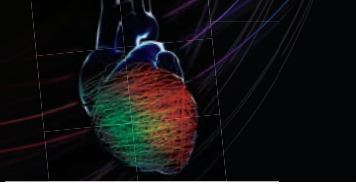
CMR being a very versatile imaging modality is capable of obtaining a 'slice' of still or moving images at any plane regardless of body habitus. Different CMR sequences, described in further detail in the next section can be employed to highlight certain types of tissue, what is termed as tissue characterisation. Additional information can be obtained with a gadolinium contrast enhanced study via perfusion dynamics as well as in a steady state 'still' contrasted image. Perfusion imaging is useful for looking at the larger vessels (aorta and pulmonary artery) and flow dynamics into myocardium or cardiac masses to demonstrate ischaemia/reduced perfusion, while the 'still' contrasted images are useful commonly in determining presence of myocardial fibrosis, infarction and presence of left ventricular thrombus (Figure 1; refer to the CMR images section).



CMR's versatility is also its weakness. And as CMR is capable of multifaceted or multiparametric imaging, it takes a longer time to complete a study. Hence a CMR study should always be directed and the indication clearly stated by the ordering physician. Its advantages and disadvantages are summarised in Table 1 below:

Table 1. Advantages and disadvantages of CMR

Advantages
• No ionising radiation involved • Imaging at any plane • No restriction by echo window • Coronary stent is not a contraindication • Gadolinium contrast use is safe in mild to moderate renal impairment • Able to differentiate between different types of tissue • Able to detect minute amounts of myocardial infarction or fibrosis (1 g or more)[4] • Ischaemia by perfusion imaging superior to single-photon emission computed tomography (SPECT)[5]
Disadvantages
• Costly • Long scan time • Breath holding required for each breath-hold • Dedicated personnel, software and hardware required • Scan space 60-70 cm wide – potential claustrophobia • Presence of irregular rhythm e.g. atrial fibrillation – unsuitable • Presence of older generation metallic surgical implants, valves, cardiac pacemakers and implantable cardioverter/defibrillators (ICDs) - unsuitable



Novel CMR technology continues to be rigorously researched and some will eventually find its way into mainstream clinical use in the near future. Some of these new techniques are summarised in Table 2 below. CMR techniques will continue to evolve rapidly and it will have tremendous potential to provide new insights to the management of cardiovascular diseases.

Table 2. Novel CMR technology

Novel Techniques	Potential Uses
T1 Mapping	Contrast free detection of diffuse fibrosis[6]
Diffusion weighted imaging	Contrast free detection of oedema, inflammation, fibrosis[7,8,9]
4D Flow	4 dimensional tracking of blood flow[10]
MR spectroscopy	Metabolic Imaging[11]
Blood oxygen level–dependent (BOLD)	Contrast free stress perfusion imaging[12]
Accelerated cine imaging	Single breath-hold acquisition of cine imaging of whole heart[13]
MR coronary angiography	Increased accuracy and resolution
Atrial scar quantification	Predicting atrial fibrillation ablation success

Section 1:
General Considerations

SECTION 1: GENERAL CONSIDERATIONS

1.1 CMR preparation, safety and contraindications

This chapter is based on the pocket guide to CMR by the European Society of Cardiology (ESC) working group,[14] Oxford Handbook of Cardiovascular Magnetic Resonance,[15] and the Society for Cardiovascular Magnetic Resonance (SCMR) website.[16]

1.1.1 GENERAL CMR CONSIDERATIONS

1.1.1.1 Preparation

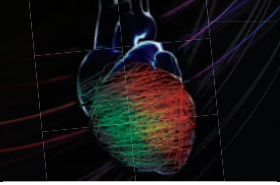
It is important to remember that the magnetic field around the MRI scanner is always switched on. Therefore, careful screening and completion of the safety screening form are mandatory.

There are no harmful biological effects of the magnetic field, however, noise levels up to 115 db during gradient switching (time varying) sequences are possible. Peripheral nerve stimulation has also been noted with the magnetic fields.

The radio frequency energy used in the formation of images is mostly transferred as heat to the patient which in turn is dependent on the thermo-regulatory mechanism of the patient. This becomes more important in patients who are diabetic, hypertensive or obese.

In optimising and preparing patients, please also consider the following, namely that the patient is able to lie flat, is in sinus rhythm, able to breath-hold, has good IV access, good placements of electrocardiogram (ECG) leads, good coil positioning and is able to wear the appropriate hospital gown.

ECG gating or triggering is mandatory in CMR and accurate triggering is crucial to obtain good quality images. Cardiac arrhythmias can pose problems to ECG gating and is a relative



contraindication to CMR. There are two types of triggering mechanisms used namely prospective and retrospective gating. The R wave on the ECG is mostly used but pulse waves or respiratory signal via the navigator sequences can also be used. Parallel imaging techniques can be used to reduce acquisition time and the use of navigator sequences is helpful if breath-hold is a problem.

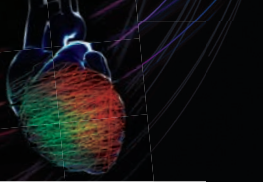
In particular, the preparation for stress CMR either with the use of adenosine or dobutamine should include the following, namely the informed consent and the patient refraining from beta blockers, theophylline, dipyridamole, nitrates, caffeine products, alcohol and cigarettes for 24-48 hours.

Most centres recommend fasting for at least 4 hours and the use of two intravenous lines is generally the standard.

1.1.2 SAFETY

When it comes to safety, it largely depends on the static magnetic field, gradient magnetic field and RF pulse which determine the specific absorption rate (SAR). A good reference would be www.mrisafety.com. This website is comprehensive and serves as a good initial reference. The practitioner would need to be familiar with the terms MR safe, MR unsafe and MR conditional devices.

Most prosthetic valves, intracoronary stents, prosthetic joints and dentures are scanned safely as are MR conditional devices. If in doubt, check the device cards, medical notes, and manufacturer's website. Most of the already implanted pacemakers and ICDs are not MR safe but there are now increasingly more MR conditional pacing systems being implanted in Malaysia.

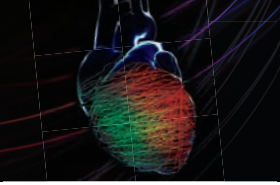


1.1.3 CONTRAINDICATIONS

Some of the more common contraindications are claustrophobia, anxiety, obesity, and severely impaired renal function.

Nephrogenic systemic fibrosis (NSF) is an independent disease involving contracture of the skin and fibrosis of the internal organs with severe consequences. It remains a rare disease and worldwide, there are about 380 reported cases. NSF was first described in 1997 and deserves special mention because of its association with the administration of gadolinium that was first postulated in 2006. This occurred especially with the older gadolinium contrast agents in patients with glomerular filtration rate (GFR) < 30 ml/min. The lifetime risk is estimated to be 1-2% in patients with end stage renal failure.

With the newer gadolinium contrasts and better patient selection in current clinical practice, the incidence of NSF has been dropping. If a contrasted CMR scan is deemed unavoidable in these patients, then the use of a non-gadodiamide/gadopentetate/gadoversetamide contrast agent is recommended with immediate dialysis thereafter to further reduce the risks of NSF.



1.2 Sequences of CMR

This section will focus on commonly used and established sequences in clinical CMR.[17] CMR protocols will be dealt in separate sections, using the SCMR standardised protocols 2013 as a guide.[18]

1.2.1 DIMENSIONS AND MORPHOLOGY

1.2.1.1 Dark blood imaging

This enables morphologic assessment of the heart, great vessels, myocardial masses and pericardium. The common sequences are spin echo techniques. Protons in non-moving or slowly moving structures (e.g. myocardium) will have high signal in the images, while rapidly flowing blood within the heart and great vessels moves out of the imaging slice, resulting in a signal void ("dark blood").

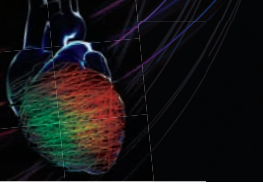
1.2.1.2 Bright blood imaging

This sequence is less sensitive to motion artifact as compared to black blood imaging. It comprises gradient echo (GRE), segmented k-space GRE, GRE hybridised with an echo-planar readout, and steady-state free precession (SSFP) techniques. Blood pool appears bright relative to the adjacent intermediate signal intensity of the myocardium. This sequence produces high temporal resolution cine movies (for ventricular function) and identifies intravoxel dephasing due to turbulent blood flow (in valvular stenosis or regurgitation).

1.2.2 MYOCARDIAL FUNCTION

1.2.2.1 Short axis and long axis cines

Newer SSFP techniques have largely been used for cine CMR assessment of myocardial volumes, mass and systolic function.



This involves intermittent 5-10 seconds breath-hold commands while obtaining consecutive short axis 6-10 mm tomographic cine short-axis cross-sections of the heart.

Long-axis views around anatomical axis of the left ventricular (LV) can also be used to assess LV function with comparable accuracy.

1.2.2.2 Single acquisition 3-dimensional method

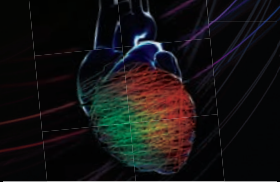
This method gives lower temporal and spatial resolution. Its primary advantage is that it only needs a single breath-hold of 20-30 seconds to cover the entire myocardium.

1.2.2.3 Myocardial tagging

Regional myocardial function may be assessed by CMR tagging. Specialised radiofrequency pulses are applied prior to the beginning of cine CMR pulses sequence. These will result in alteration of the magnetic properties of the heart ("dark grid stripe pattern"). The grids will be displaced by myocardial contraction. It is usually interpreted qualitatively, with specialised software for the calculation of regional myocardial strain for research purposes.

1.2.3 PHASE-CONTRAST (PC) BLOOD FLOW

Blood flowing through a magnetic field gradient produces a phase shift that is proportional to the velocity of flow. By summing the PC-generated velocities within the area of the lumen throughout the cardiac cycle, blood flow within the vessel can be calculated. This method strongly agrees with results obtained in phantom models, non-invasive and other accepted invasive techniques.



1.2.4 MYOCARDIAL PERFUSION

The commonest sequence used is rapid dynamic imaging during the first pass of contrast agent.

Gadolinium-based contrast agents in CMR reduce both the longitudinal (T1) and transverse (T2) relaxation times. Pulse sequence techniques sensitive to T1, T2, or both are employed to detect the transit of contrast agent through a myocardial perfusion bed. Currently, myocardial perfusion studies are mostly based on T1-weighted 2D, multislice imaging, with 3 to 5 slices coverage of the heart.

Microvascular dysfunction and microvascular obstruction (MVO) after myocardial infarction can also be detected with this sequence.

1.2.5 ANGIOGRAPHY

MR angiography (MRA) image acquisitions are typically 3D. Image post processing through maximum intensity projection (MIP) and multiplanar reformation (MPR) then enables assessment of complex geometries of carotid arteries, aorta, renal arteries, and peripheral vasculature.

Conventional T1- and T2-weighted dark blood techniques enable proper depiction of vessel walls. Bright blood imaging techniques (time-of-flight, phase contrast, SSFP and contrast-enhanced MRA) enable evaluation of blood flow and vessel lumens that allow 3D display of vascular anatomy.

Time-resolved MRA (rapid frame rate MRA) allows direct visualisation of flow dynamics, which may be used for assessment of vascular shunts or dissections.

1.2.6 TISSUE CHARACTERISATION

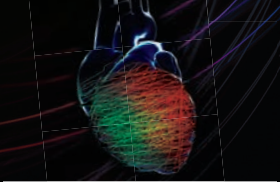
This sequence uses characteristics of proton relaxation (T1, T2, and T2* relaxation times). T1 images are often used for contrast-enhanced studies. T2 and T2* imaging are mostly used in non-contrast approaches. Among the commonly used non-contrast sequences in clinical CMR are T2-weighted imaging to assess myocardial water content and T2* imaging to assess myocardial iron overload.

Gadolinium (Gd) chelates shorten the T1 relaxation time within the surrounding tissue. Regions with high Gd concentration during T1-weighted imaging will have increased signal intensity. Gd chelates facilitate water visualisation in the intravascular (blood) or in the extravascular organ tissue space. This can be used to selectively identify areas with reduced or increased "uptake" of Gd.

This concept can be used during the "first-pass imaging" as mentioned before and also during the delayed enhancement imaging. Several minutes after intravenous administration of Gd, larger volume of distribution will be available in necrotic or fibrotic myocardium resulting in a higher concentration of contrast agent (Figures 3B and 3C). This is referred to as "delayed hyperenhancement" or "late gadolinium enhancement" (LGE).

1.2.7 METABOLISM

Cardiac magnetic resonance spectroscopy (MRS) allows assessment of myocardial metabolism without radiation or contrast agents. It uses the intrinsic magnetic resonance signals from nuclei, including (31)Phosphorus, (1)Hydrogen, (23)Sodium, and (13)Carbon and also hyperpolarisation techniques. However, currently MRS is limited to research applications due to its low temporal and spatial resolution.



1.3 Drugs and contrasts used in CMR

This chapter is based on the updated standardised SCMR Protocols 2013[18] and Medscape.[19]

Most of the CMR procedures use intravenous gadolinium-based contrast agents and pharmacologic stress agents for myocardial perfusion assessments. Table 3 lists the stress agents and their pharmacological actions.

Table 3. Stress agents and their pharmacological actions

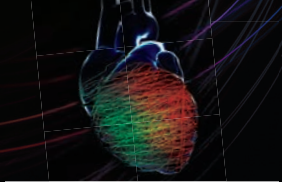
Stress agents	Mode of action	Dose
Dobutamine	A synthetic catecholamine, which directly stimulates predominantly beta-1 and mildly beta-2 receptors, causing increase in heart rate, blood pressure, and myocardial contractility.	10-40 ug/kg/min*
Atropine	Competitive antagonist of muscarinic acetylcholine receptors hence producing chronotropic effect.	0.25-2 mg*
Adenosine	A non-selective A2A adenosine receptor agonist, direct vasodilator effect causing up to 4 fold increased blood flow in normal vessels. Blunted reduction of response in stenotic vessels cause relative decreased perfusion.	140-210 ug/kg/min**
Regadenoson	A selective A2A adenosine receptor agonist, direct vasodilator effect increasing coronary blood flow similar to adenosine. Longer half-life allows for a bolus dose.	0.4 mg single injection***

* *To achieve 85-100% of maximum predicted heart rate*
** *Consider up-titration if after 2-3 minutes, heart rate does not increase by 10 bpm and/or blood pressure does not drop by > 10 mmHg*
*** *Not currently available in Malaysia. It is an attractive single bolus administration alternative to adenosine infusion.*

1.3.1 CONTRAINDICATIONS AND SIDE EFFECTS

Table 4. Stress agents and their contraindications and side effects

Stress agents	Contraindications	Side effects
Dobutamine	• Severe systemic arterial hypertension (≥ 220/120 mmHg) • Unstable angina pectoris • Significant aortic valve stenosis (Peak aortic valve gradient > 50 mmHg or aortic valve area < 1 cm^2) • Complex cardiac arrhythmias including uncontrolled atrial fibrillation • Hypertrophic obstructive cardiomyopathy (HOCM) • Myocarditis, endocarditis, pericarditis • Uncontrolled congestive heart failure	• Dobutamine at high doses may cause chest pain and/or palpitations. • More severe complications are rare, including: » infarction » ventricular fibrillation » sustained ventricular tachycardia
Adenosine or Regadenoson	• 2nd or 3rd degree atrioventricular (AV) block or sinus node dysfunction • Systolic blood pressure less than 90 mmHg • Sinus bradycardia (heart rate < 40 bpm) • Active bronchoconstrictive or bronchospastic disease with regular use of inhalers • Known hypersensitivity to adenosine or regadenoson **Note:** Side effects are described as less significant with regadenoson than with adenosine, however, the half-life of regadenoson is longer.	• May cause flushing, chest pain, palpitations and/or breathlessness. • More severe side effects include: » transient heart block » transient hypotension » transient sinus tachycardia » bronchospasm
Atropine	• Narrow-angle glaucoma • Myasthenia gravis • Obstructive uropathy • Obstructive gastrointestinal disorders	



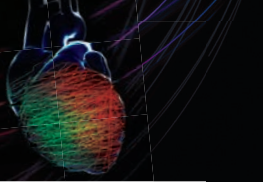
1.3.1.1 Gadolinium dosing module/safety

Table 5. Suggested dose for contrast and saline chasing bolus and injection rates.

Indication	Contrast dose (mmol/kg)	Injection rate	Saline chasing bolus	Injection rate
Perfusion	0.05-0.1	3-7 ml/s	30 ml	3-7 ml/s
Late gadolinium enhancement (LGE)	0.1-0.2	-	20 ml	-
Angiography (carotids, renals, aorta)	0.1-0.2	2-3 ml/s	20 ml	2-3 ml/s

1.3.1.2 Safety considerations for gadolinium-based contrast

The use of gadolinium contrast should be avoided in patients with stage 4 or 5 chronic kidney disease (eGFR < 30 mL/min/1.73m^2), particularly for those on dialysis, as well as patients with acute renal failure and chronic liver disease, due to concerns regarding NSF. The dose of gadolinium contrast used should be as low as possible to achieve adequate image quality. If no alternative is available in dialysis patients such that gadolinium must be used, dialysis should be performed as per institutional, regional or national guidelines. Further details on safety are elaborated in the CMR preparation, safety and contraindications chapter.



1.4 Guidelines for reporting CMR studies

This consensus on CMR reporting draws on previously published documents from professional societies (SCMR, American College of Cardiology Foundation, American Heart Association, American College of Radiology, and North American Society for Cardiovascular Imaging) and is tailored to provide a framework to report CMR studies within the context of the Malaysian healthcare system.[17, 20, 21] It is reasonable to differ from the recommendation at the discretion of the reporting physician in consideration of the circumstances of each individual patient, available facility and any new developments in knowledge and technology following the publication of this consensus document.

This background information pertaining to the CMR examination is recommended to be recorded which may or may not be included in the final report:

1) Healthcare facility identity

2) Patient demographics
   a) Name
   b) Gender
   c) Age
   d) Identification number/medical record number
   e) Date of birth
   f) Weight, height and body surface area

3) Study parameters
   a) Referring physician/referring hospital
   b) Scan date
   c) Indication for study
   d) Background medical history and other relevant patient information
   e) Staff involved in the procedure
   f) Listing of sequences used – optional
   g) Quality of images acquired

SECTION 1: GENERAL CONSIDERATIONS

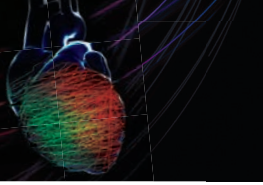
The following CMR study findings wherever relevant to the study indication are recommended to be included in the final report:

1) Left and right ventricular structure and function
   a) Ventricular volumes* – end diastolic and end systolic volumes
   b) Ejection fraction
   c) Ventricular mass*
   d) Regional wall motion abnormalities – according to the ACC/AHA 17-segment myocardial model (Figure A)
   e) Wall thickness – end diastolic

   * Adjustment for body size recommended by dividing raw measures with body surface area to obtain indexed values

2) Valves (see section on valvular heart disease assessment)
   a) Morphology of valve complex (leaflets, annulus, chordae)
   b) Presence of any insufficiency or reduced valvular excursion
   c) Regurgitant volumes and fraction
   d) Peak velocities
   e) Valve area determination by planimetry

3) Aorta
   a) Measurement of dimensions, inner diameter (lumen width) in end diastole
      i. Aortic annulus
      ii. Sinuses of valsalva
      iii. Sinotubular junction
      iv. Ascending aorta at the level of pulmonary artery
      v. Proximal aortic arch
      vi. Mid aortic arch
      vii. Distal aortic arch
      viii. Descending aorta at the level of pulmonary artery
      ix. Descending aorta at the level of diaphragm



b) Description of aortic atherosclerosis
c) Aortic aneurysm
    i. Size
    ii. Morphology (saccular, fusiform)
    iii. Location, relation to branch vessels and adjacent structures
    iv. Mural thrombus if present
d) Aortic dissection
    i. Dissection classification (Debakey or Stanford)
    ii. Location of tear or areas of communication
    iii. Size and extent of true and false lumen
    iv. Presence of mural thrombus
    v. Branch vessel involvement
    vi. Presence of periaortic, mediastinal, pericardial or pleural effusion

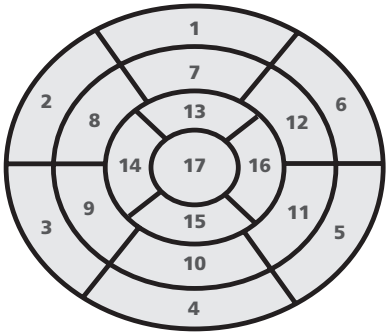
4) Late gadolinium enhancement (LGE)
    a) Location – according to the ACC/AHA 17-segment myocardial model (Figure A)
    b) Pattern of enhancement – subepicardial, intramural/mid-wall, subendocardial, transmural
    c) Transmural extent defined as
        i. ≤ 25%
        ii. 26 – 50%
        iii. 51 – 75%
        iv. ≥ 75%

5) Myocardial perfusion
    a) Microvascular obstruction/dysfunction if present
    b) Flow deficits indicative of myocardial ischaemia
        i. Location – according to the ACC/AHA 17-segment myocardial model (Figure A)
        ii. Extent and severity
        iii. Vessel territory

6) Congenital heart disease
   a) Situs
   b) Ventriculoarterial
   c) Atrioventricular relationship
   d) Pulmonary venous connections
   e) Systemic veins and connections
   f) Septal defects
   g) Valvular lesions
   h) Pulmonary arteries
   i) Aorta
   j) Presence of shunt or conduit

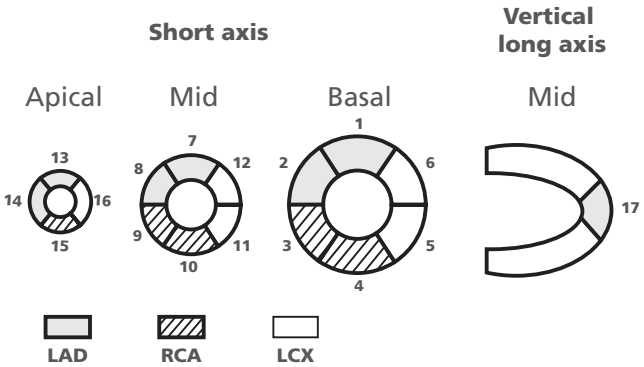
Figure A.
AHA/ACC 17 segment myocardial model and coronary artery territories

Left ventricular segmentation



1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex

Coronary Artery Territories

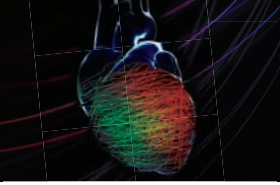


Reproduced with permission from Hundley WG, Bluemke D, Bogaert JG, Friedrich MG, Higgins CB, Lawson MA, *et al.* Society for Cardiovascular Magnetic Resonance guidelines for reporting cardiovascular magnetic resonance examinations. *J Cardiovasc Magn Reson.* 2009;11:5.[20]

7) Extracardiac finding
   It is recognised that the sequences, field of view and techniques in CMR are not designed to delineate extra-cardiac structures. Any incidental findings may be reported and further imaging modalities recommended for confirmation.

8) Summary and conclusion
   The final written report should include a concluding summary statement relating to the study indication, signature of reporting personnel and date of report.

Communication of findings to patient's managing physician is of utmost importance and should be performed in a timely manner within the capacity of available resources and manpower. The final written report should be stored and archived within the healthcare facility.



1.5 Training and competence

With the growing popularity of CMR, there is a need to ensure a certain level of competency is maintained in CMR acquisition and reporting. These training and competency recommendations are tailored to the current Malaysian setting where CMR is still a new field, and will be subjected to a review in the future. CMR training is divided into levels 1, 2 and 3.

All specialists who perform and report CMR examinations should have knowledge of the basics of CMR imaging and their indications.

General criteria needed to be fulfilled prior to embarking on CMR training includes:

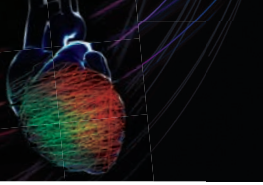
1. Registered with the Malaysian Medical Council.
2. Holds a current Annual Practicing Certificate.
3. Board certification (National Specialist Registry) in Cardiology, Clinical Radiology or Nuclear Medicine.
4. Basic knowledge, clinical training and experience in at least one other cardiovascular imaging modality.

1.5.1 LEVEL 1: GENERAL TRAINING

Upon completion of training, the trainee will have adequate knowledge on the background of CMR and the indications for which this examination is used, but not for the practice or interpretation of CMR examinations independently.

Training requirements include:

1. Exposure to the methods and multiple applications of CMR.



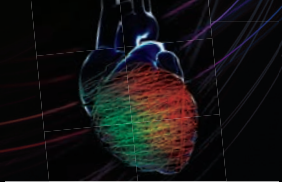
2. Mentored (with a Level 2 or Level 3-trained physician-mentor) interpretation of at least 30 CMR examinations, which consist of a wide variety of conditions. These examinations may be obtained from an established CMR-teaching file.
3. Attend didactic lectures on the basics of CMR, and parallel teaching material on the subject. Within this material, are also information that provides the trainee a basic understanding of magnetic resonance physics.
4. Provide proof of training (e.g. verified log book, letter of certification by a qualified level 2 or 3 mentor or equivalent*.

1.5.2 LEVEL 2: SPECIALISED TRAINING

Training for Level 2 should include Level 1 CMR training experience. Trainees who have completed Level 2 training recommendations are those who seek to practise the clinical subspecialty of CMR, and interpretation of CMR examinations. Level 2 trainees must have a minimum of 3 months of dedicated CMR training, whereby 1 month is defined as 4 weeks, and 1 week defined as 35 hours. During the course of the 3 month training, the trainee must be under the supervision of a Level 2 or Level 3 (preferred) mentor.

Training requirements include:

1. Mentored (with a Level 2 or Level 3-trained physician-mentor) interpretation of at least 100 CMR examinations, in which at least 30 should be when the trainee is present during the scanning procedure, ideally as the primary operator, and is the primary interpreter. Substantial proportions (at least 15) must include cardiac pharmacological stress studies. The rest of the examinations can be obtained from an established study file, archived studies, online materials or other historical literature source.



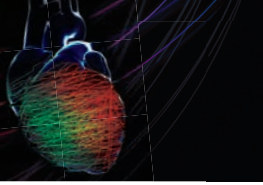
2. Familiarity with associated specialised CMR techniques such as pharmacological stress, CMR-specific contrast agents, electrocardiographic/pulse gating and also post-processing software.
3. Attend coursework and didactic lectures (in greater depth than in Level 1). Topics on CMR physics and applications, safety, indications, interpretations and contraindications of contemporary CMR should be included.
4. Provide proof of training (e.g. verified log book, letter of certification by a qualified level 2 or 3 mentor or equivalent*.

1.5.3 LEVEL 3: ADVANCED TRAINING

Training for Level 3 represents the highest level of training, whereby the trainee undertakes a minimum of 12 months intensive training under the supervision of a Level 3 mentor, with the intention of pursuing a clinical or academic career in CMR or directing a CMR laboratory.

Training requirements include:

1. Supervised clinical imaging and interpretation of at least 300 CMR examinations. The trainee must be physically present where 100 of those examinations are undertaken. In the remaining 200 cases, at least 100 of these should be reviewed by the mentor, and the remaining cases can be from an established training folder, archived studies, online materials or other historical literature source.
2. Obtained in-depth knowledge beyond Level 2 through coursework and didactic lectures, particularly in the subjects of CMR physics, CMR applications and post-processing software, such that the trainee may improve the diagnostic capacity of this evolving technology.



3. Participate in CMR basic or clinical research, with a primary role in a specific area of that research.
4. Provide proof of training (e.g. verified log book, letter of attestation by a Level 3 mentor or equivalent**).

1.5.4 MAINTENANCE OF COMPETENCY

As per any other procedure, a minimum of cases are required to be undertaken by the specialists to maintain continued proficiency in quality of care.

Maintenance of CMR expertise requires both (1) clinical cases to be undertaken and interpreted, and (2) continuing medical education (CME) in CMR or associated fields in cardiac imaging.

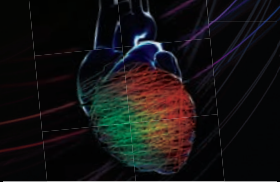
Criteria for maintenance of competency include:

1. A minimum of 30 CMR examinations are seen annually. However, in circumstances where this is not possible, alternative measures to monitor and evaluate the specialist who undertake CMR examinations and interpretation can be considered.
2. A total of 20 hours of CME in CMR over two years.

* Definition of a Level 2 mentor or equivalent: 1) A specialist certified/recognised by a reputable international CMR body; or 2) A specialist fulfilling the Level 2 requirement with at least 2 years of reporting CMR independently, and credentialed by his/her own institution.

** Definition of a Level 3 mentor or equivalent: 1) A specialist certified/recognised by a reputable international CMR body; or 2) A specialist fulfilling the Level 3 requirement with at least 1 year of reporting CMR independently, and credentialed by his/her own institution.

The section above was based on the SCMR guidelines,[22] Pohost GM, *et al* 2008,[23] Budoff MH 2005,[24] and Kramer CM 2007.[25]



1.5.5 RADIOGRAPHERS[26]

All CMR scanners in Malaysia should be operated by qualified personnel. Criteria for a qualified personnel include:

1. Radiographers with a diploma or degree in radiography, or any equivalent radiography qualifications recognised by the Ministry of Health Malaysia or Society of Radiographers Malaysia.
2. Obtained advanced certification in at least post basic MR. If the radiographer does not have an advance certification in MR, a minimum of 3 months full time MR training is required under the supervision of a suitably trained MR radiographer and radiologist before being allowed to operate an MR scanner independently. Radiographers are encouraged to keep a logbook of the number of CMR cases performed.
3. Ability to prepare, position, ensure patient safety, monitor the patient, apply the contrast injection and scanning protocol as prescribed by the supervising doctors.
4. Perform regular quality control testing.
5. Adequate Continuous Professional Development on CMR related topics (at least attend one conference/ workshop/course every 2 years).

MR scanners should not be operated by any person without the above stated qualifications e.g. medical physicists, technicians, research staff, post doctorate fellows, nurses and any other non-radiological qualified staff.

Intravenous contrast materials can be administered by radiographers and nurses under the direction of supervising doctors, if the practice is in compliance with institutional regulations.

CMR Images

Ischaemic cardiomyopathy

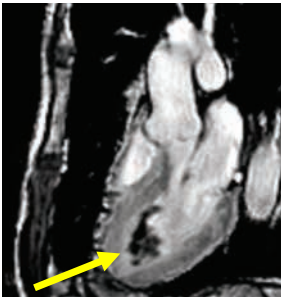


Figure 1.
LV thrombus in the apex in the EGE sequence.

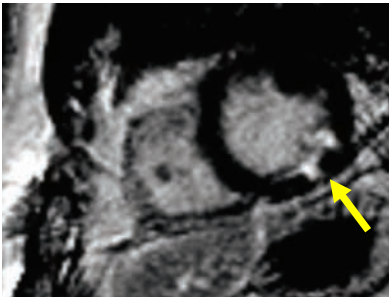


Figure 2.
Partial thickness or subendocardial infarction in the inferior wall.

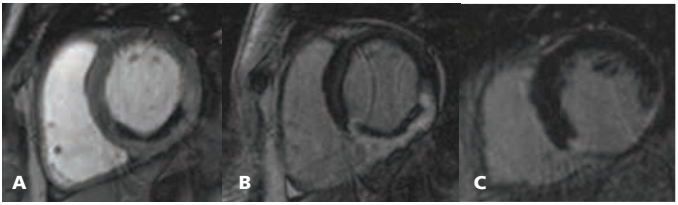


Figure 3.
(A) Microvascular obstruction (MVO) in the EGE sequence in acute inferior myocardial infarction. **(B)** LGE sequence shows full thickness hyperenhancement with MVO. **(C)** LGE sequence in chronic phase showing resolution of MVO.

CMR stress perfusion imaging

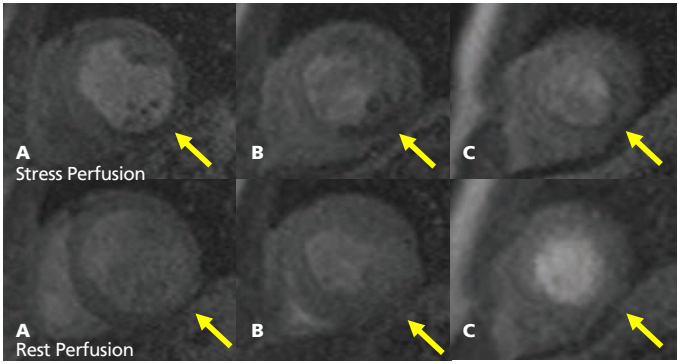


Figure 4.
Stress and rest adenosine perfusion Imaging at the basal **(A)**, mid **(B)**, and apical **(C)** levels demonstrating reversible perfusion defect at the right coronary artery territory.

Myocarditis

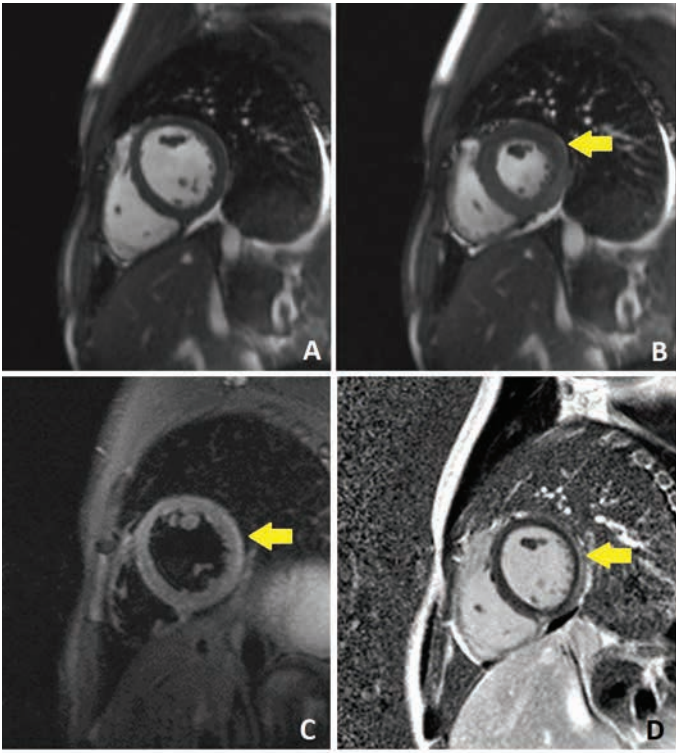


Figure 5.
CMR images showing presence of active myocarditis. **(A)** and **(B)** show short axis cine MRI view of the left ventricle with subtle hypokinesis of the lateral walls. **(C)** shows a T2-STIR image depicting oedema of the subepicardial lateral and inferolateral left ventricular wall. **(D)** shows the LGE image depicting subepicardial uptake in the inferolateral region of the left ventricular wall.

Familial hypertrophic cardiomyopathy

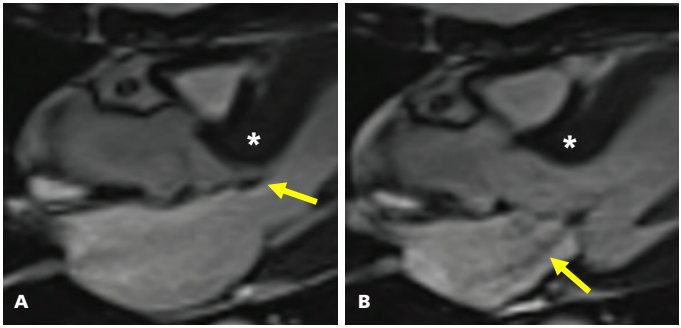


Figure 6.
Cine SSFP 3 chamber view shows asymmetrical septal hypertrophy (asterisks) with narrow left ventricular outflow tract due to systolic anterior motion of the anterior leaflet of the mitral valve (arrow). Note associated mitral regurgitation (arrowhead).

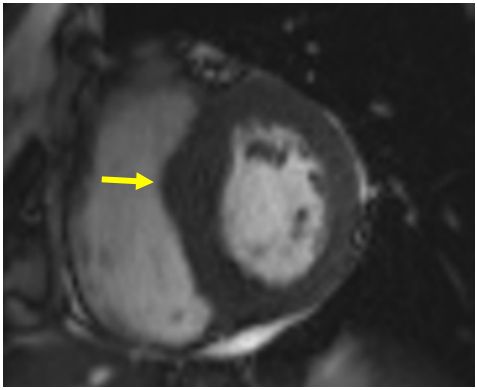


Figure 7.
Asymmetric hypertrophic cardiomyopathy involving the septum. The short axis view demonstrating septal thickening (arrow).

Iron-loading conditions

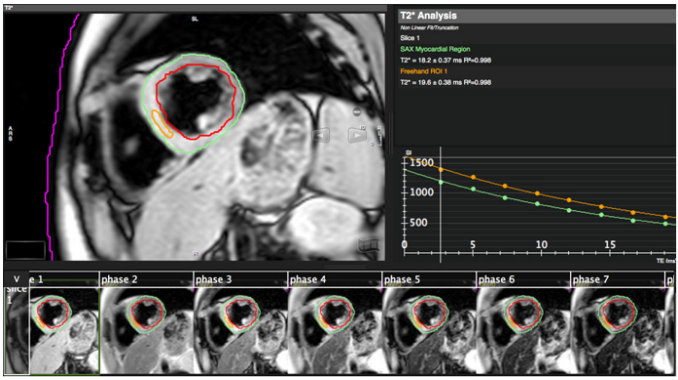


Figure 8.
Cardiac iron overload in thalassemia.

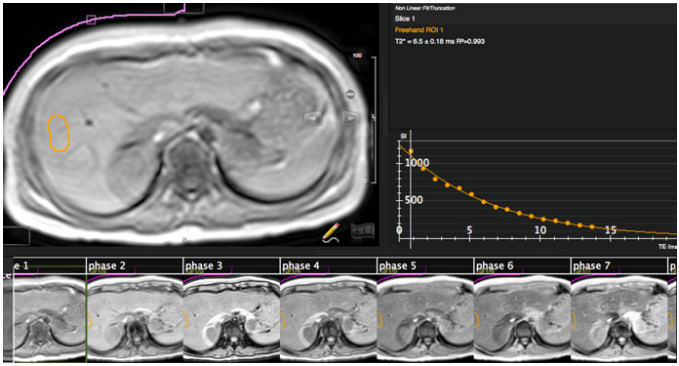


Figure 9.
T2* assessment of the liver in the same patient showed normal T2* level.

Amyloidosis

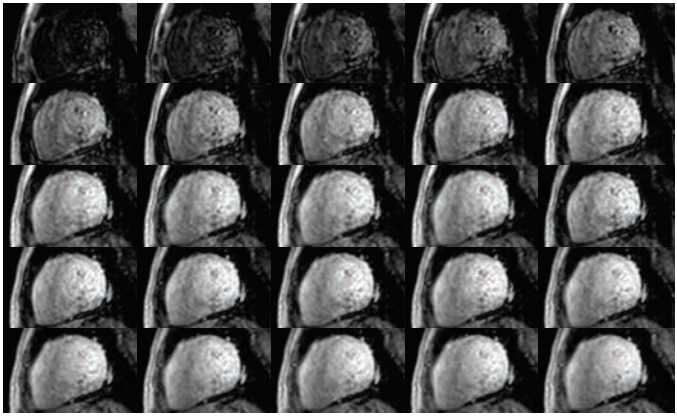


Figure 10.
Poor nulling in T1 scout sequence is characteristic of amyloidosis.

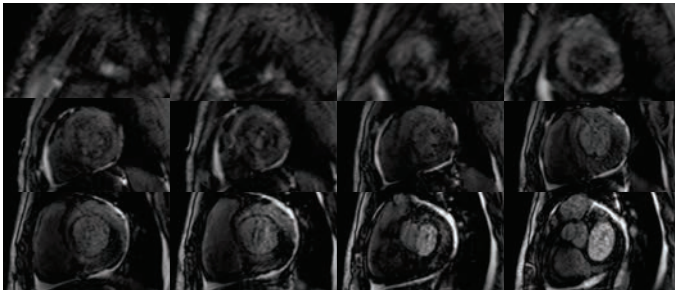


Figure 11.
LGE sequence shows diffuse involvement with poor blood pool/ myocardium differentiation due to amyloid infiltration.

Sarcoidosis

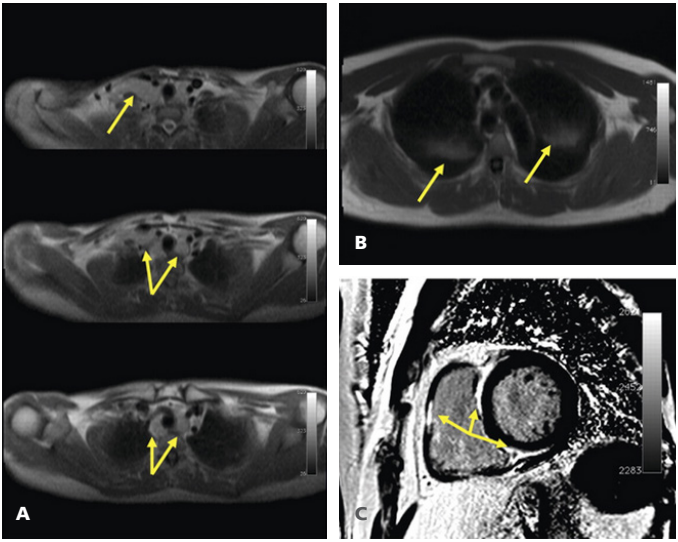


Figure 12.
Images of three patients with sarcoidosis. **(A)** HASTE images from the upper thorax demonstrate right supraclavicular and bilateral upper paratracheal lymphadenopathy (yellow arrows). **(B)** Axial HASTE image at the level of the upper aortic arch demonstrating bilateral pulmonary parenchymal high signal within the posterior upper lobes (yellow arrows). **(C)** Phase-sensitive inversion-recovery delayed gadolinium image shows multiple areas of localised enhancement (yellow arrows) affecting both ventricles in sarcoidosis.

Left ventricular non-compaction

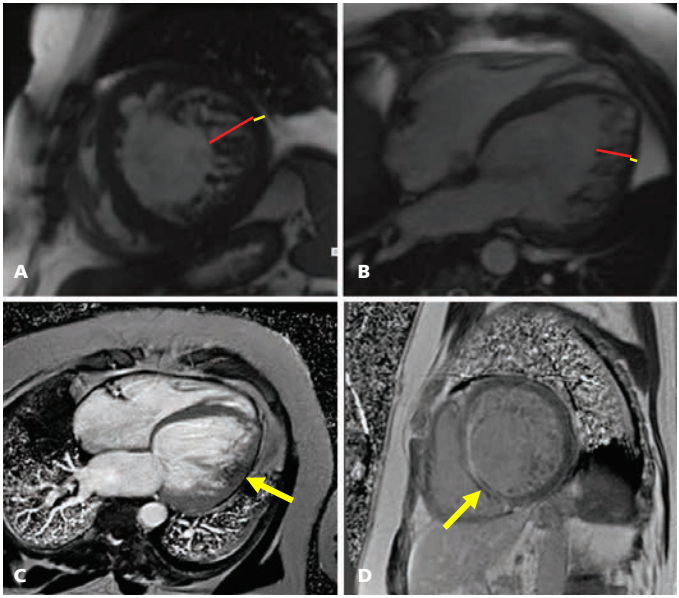


Figure 13.
CMR images showing left ventricular non-compaction. **(A)** and **(B)** show mid-ventricular short axis (SA) and horizontal long axis (HLA) views detailing excellent trabecular to blood contrast with an NC:C ratio of > 2.3 (red line NC, yellow line C). Figure **(C)** shows EGE image with hypoenhanced material adjacent to the subendocardial, compacted region which is consistent with a thrombus. **(D)** shows LGE with mid-wall hyperenhancement signifying myocardial fibrosis.

Tako-Tsubo cardiomyopathy

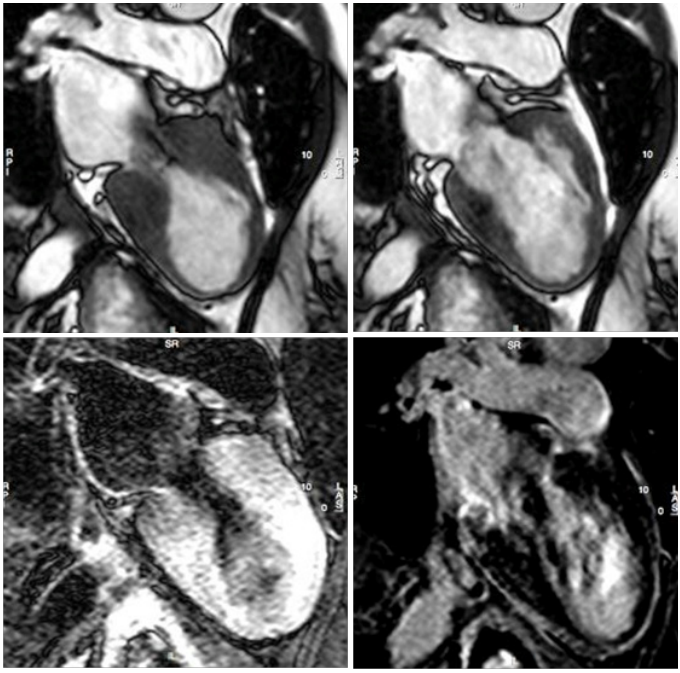


Figure 14.
Cine of 2 chamber (2CH) views in Systole and Diastole (1st panel). 2nd panel shows increased signal intensity of the corresponding RWMA in T2STIR sequence, but without any evidence of late gadolinium enhancement.

Congenital heart disease

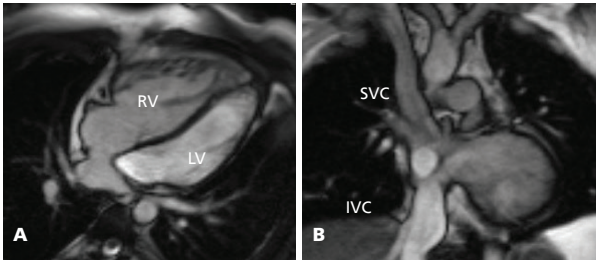


Figure 15.
Transposition of great arteries post atrial switch. **(A)** The pulmonary veins are baffled into the morphological right ventricle while **(B)** the systemic veins are baffled into the morphological left ventricle. CMR is a useful tool to interrogate baffle obstruction which uncommonly occurs in patients post atrial switch. RV : right ventricle; LV : left ventricle; RA : right atrium; SVC : superior vena cava; IVC : inferior vena cava.

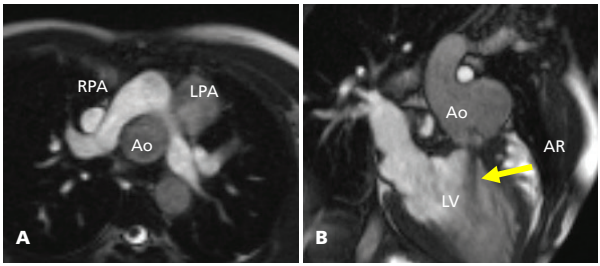


Figure 16.
Transposition of great arteries post arterial switch. **(A)** Assessment of the branch pulmonary arteries post Le Compte procedure to rule out branch pulmonary artery stenosis. If stenosis is present, through plane flow of the branch pulmonary arteries allows for the interrogation of the split pulmonary arterial flow. **(B)** Dilated aortic root, a common complication post surgery is assessed on MRI. * RPA: right pulmonary artery; LPA : left pulmonary artery; Ao : aorta; AR : aortic regurgitation; LV : left ventricle.

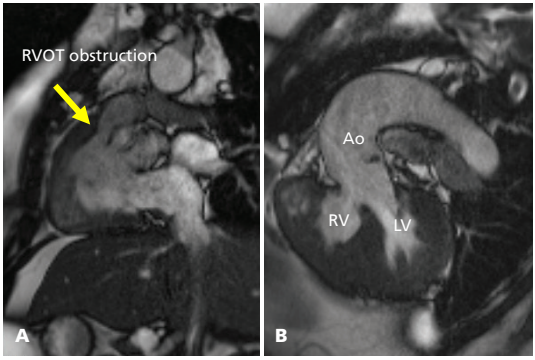


Figure 17.
Preoperative assessment of a patient with tetralogy of Fallot showed **(A)** subvalve pulmonary stenosis with good size pulmonary valve and **(B)** overriding of the aorta. RVOT: right ventricular outflow tract; RV : right ventricle; LV : left ventricle; Ao : aorta.

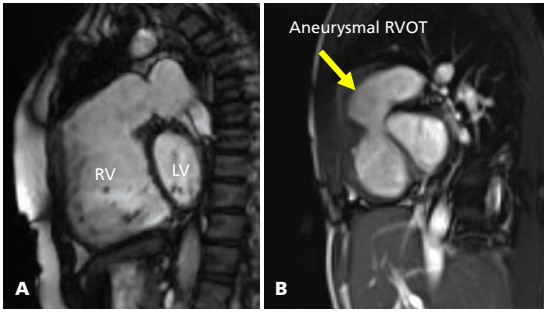


Figure 18.
Tetralogy of Fallot post repair. **(A)** Quantification of RV dimension and systolic function are important to determine the optimal timing for pulmonary valve replacement. **(B)** Aneurysmal right ventricular outflow tract (RVOT) following ventriculotomy during the initial repair needs to be identified and corrected. RV : right ventricle; LV : left ventricle.

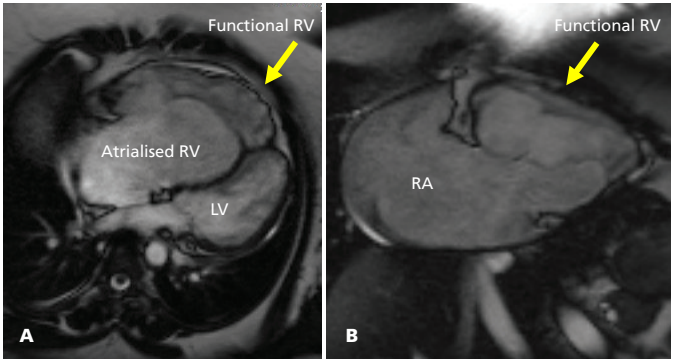


Figure 19.
Ebstein's anomaly. **(A)** CMR is helpful in identifying the portion of atrialised right ventricle and the functional right ventricle. **(B)** The tricuspid valve is deviated towards the right ventricular outflow tract. RV : right ventricle; LV : left ventricle; RA : right atrium.

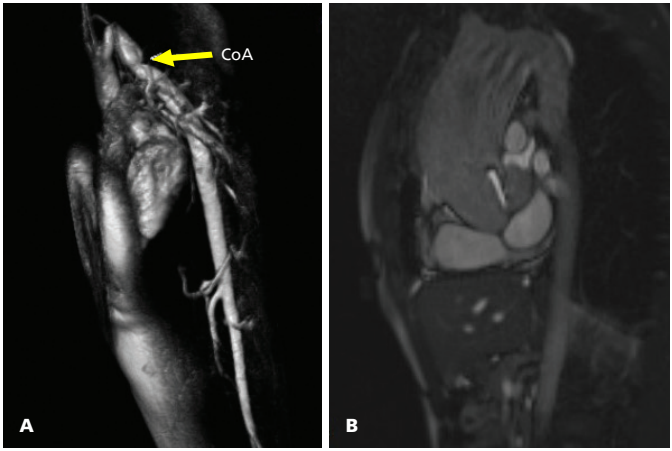


Figure 20.
CMR allows for assessment of the whole arch. **(A)** Coarctation of aorta. **(B)** Severely dilated ascending aorta in a patient with bicuspid aorta valve.

CMR in cardiac masses and pericardial disease

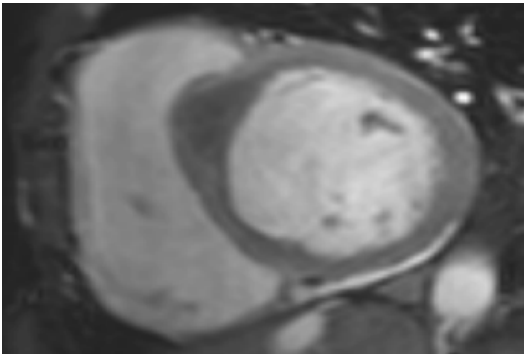


Figure 21.
Benign cardiac mass (fibroma).

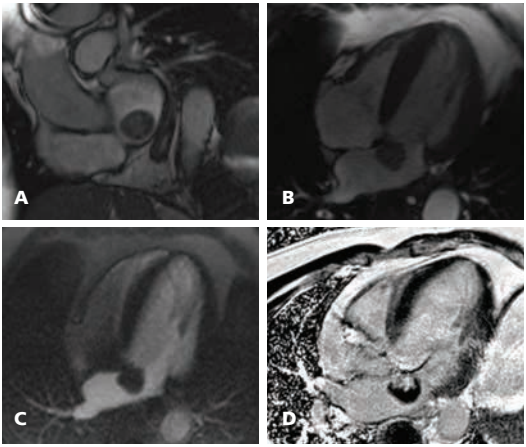


Figure 22.
LA myxoma in the short axis **(A)** and 4 chamber cine views **(B)**. Typically there is no perfusion seen in the first pass imaging **(C)** and there is presence of patchy hyperenhancement in the LGE sequence **(D)**.

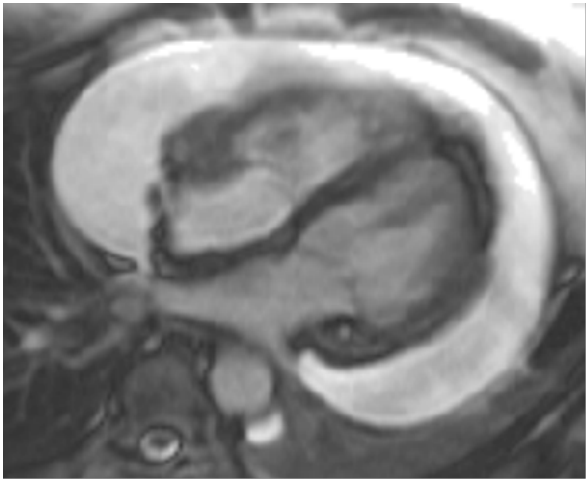


Figure 23.
Metastasis and malignant pericardial effusion.

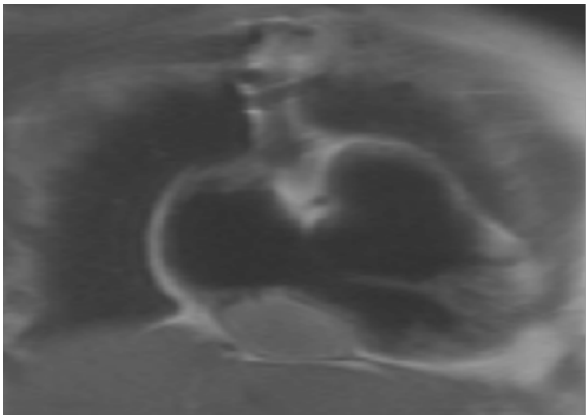


Figure 24.
Pericardial cyst.

Extra cardiac findings in CMR

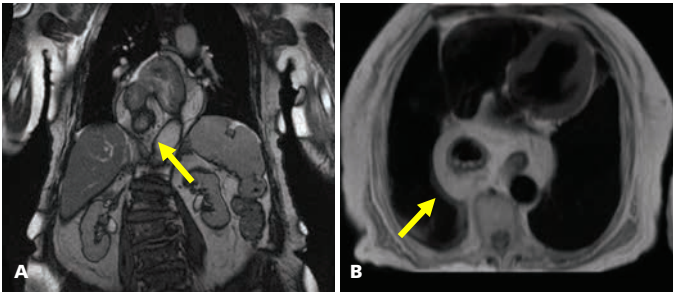


Figure 25.
Coronal scout image demonstrating an abdominal mass in continuity with the thoracic cavity **(A)** and short axis HASTE image demonstrating a hollow viscus in the thoracic cavity consistent with a hiatus hernia.

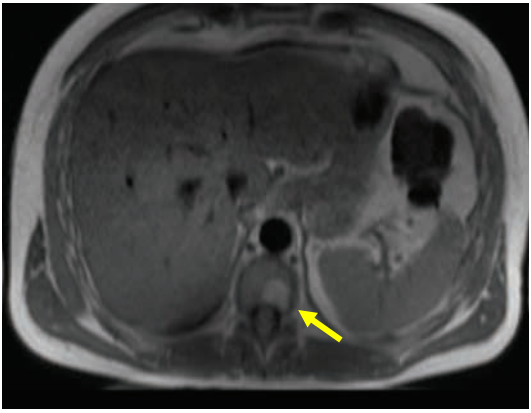


Figure 26.
Lower thoracic HASTE sequence demonstrating isolated rounded high signal vertegral body lesion judged to reflect a vertebral body haemangioma.

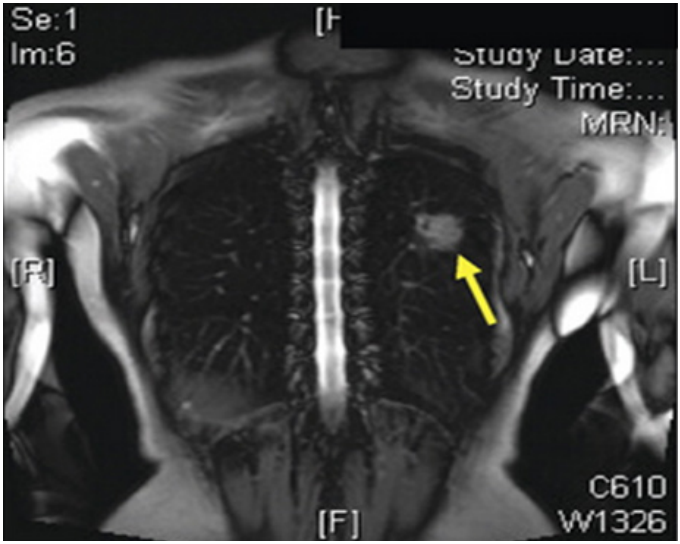
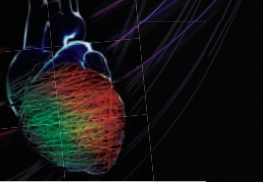


Figure 27.
A 43-year-old female non-smoker referred for evaluation of atypical chest pains. Coronal localiser depicts an ill-defined region of increased signal in the apical segment of the left lower lobe. Further investigations later confirmed this is a lung carcinoma.

Section 2: Indications



2.1 CMR in coronary artery disease

2.1.1 OVERVIEW

CMR offers a comprehensive assessment of patients with coronary artery disease in that it is multiparametric and uses different CMR sequences to assess cardiac anatomy, ventricular function, myocardial perfusion and infarction which traditionally mandated multimodality imaging.

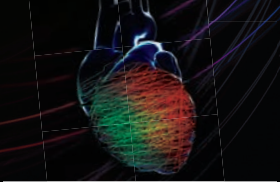
Being very sensitive in detecting acute and chronic infarction, it is superior to SPECT[27] and when compared to ECG has up to 390% higher rate of detection.[28] Infarcted myocardium as little as 1 g can be detected[4] compared to the minimum of 10 g in SPECT.[29] Associated structural complications of myocardial infarction such as left ventricular clot, aneurysmal dilatation, and rupture of interventricular septum are well demonstrated as well.

Non-ischaemic conditions like *Tako-tsubo*, myocarditis, trauma, pulmonary embolism or drug toxicities that present similarly with acute coronary syndrome with the triad of chest pain, dynamic ECG changes and raised cardiac enzymes can easily be differentiated with CMR assessment.[28]

Comprehensive CMR assessment includes:

A) Ventricular wall thickness and contractile dysfunction:
   - Wall thickness and regional wall motion abnormalities similar to echocardiography.

B) Oedematous myocardium, in acute coronary syndrome:
   - Areas of acute injury (oedema) can be identified.
   - The area of myocardial oedema extending beyond the area of infarction (area at risk) can be identified and quantified. It carries prognostic implications and its clinical value is actively being researched.[30]



C) Inducible ischaemia:
- A vasodilator stress, commonly adenosine is used to unmask areas of poor perfusion.
- Areas of poorly perfused myocardium during stress indicate reversible ischaemia from a functionally significant coronary artery stenosis.
- Technique is superior to SPECT.[31]

D) Infarction detection or viability imaging:
- LGE imaging is the gold standard for in vivo infarct imaging.[31]
- Prognostic value in predicting revascularisation outcomes.[32]

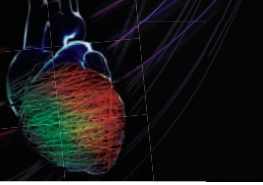
E) Microvascular obstruction (Figures 3A and 3B), acute coronary syndrome:
- Its presence indicates a poor prognosticator.[33]

F) Left ventricular thrombus imaging (Figure 1):
- Superior to echocardiography due to better morphological detection and tissue characterisation capability.[34]

G) Coronary artery anatomy imaging:
- Covered under MR Angiography and MR Coronary Angiography Chapter.

2.1.2 ISCHAEMIC CARDIOMYOPATHY

Ischaemic cardiomyopathy is usually diagnosed following a clinical presentation of acute/chronic heart failure or may be diagnosed in patients presenting with coronary artery disease. The diagnosis is generally confirmed by means of echocardiography which demonstrates features such as ventricular dilatation, impaired ejection fraction and myocardial wall motion abnormalities.



In Malaysia, CMR currently plays an adjunctive role in the assessment of patients with ischaemic cardiomyopathy, providing additional information to the treating physician to enable evidenced-based clinical decisions.

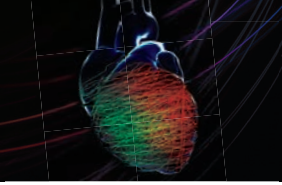
The roles of CMR in the assessment and management of ischaemic cardiomyopathy are as follows:

2.1.2.1 Assessment of ventricular volumes, ejection fraction and mass

As mentioned previously, in comparison to echocardiography, CMR imaging provides excellent visualisation of myocardial tissues due to a higher spatial resolution and the ability to obtain images in any desired plane without acoustic window and operator dependent limitations.

Furthermore, echocardiographic measurements of left ventricular volumes and function are based on geometrical assumptions that do not apply in the asymmetrically remodelled cardiomyopathic heart.[35] CMR assessment is independent of these geometrical assumptions and is able to provide reproducible and reliable measurements without exposure to ionising radiation.[35]

For these similar reason, CMR has been able to demonstrate validated and reproducible measurements of volumes, function and mass of the complex structured right ventricle (RV).[36-38] As a result of these factors, when compared to echocardiography and radionuclide ventriculography, CMR is the preferred modality to assess ejection fraction, ventricular volumes and mass in patients with heart failure.[35,39]



2.1.2.2 Assessment of myocardial viability

Dysfunctional myocardium in ischaemic cardiomyopathy may be indicative of either non-viable scar tissue or viable but dysfunctional tissue (hibernation or stunning). Revascularisation of viable myocardial tissue in patients with ischaemic cardiomyopathy is associated with improved systolic function, symptoms and survival.[40]

2.1.2.2.1 Assessment of regional myocardial thickness

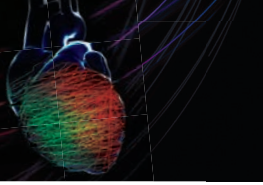
It is conventionally accepted that myocardial thickness of less than 5.5 mm in echocardiography equates to non-viability and this is similar in CMR.[41,42] However, recent studies have suggested that myocardial thickness of less than 5 mm may be viable especially if there is no LGE.[43,44]

2.1.2.2.2 Assessment of regional contractile reserve with dobutamine stress

CMR is capable of wall motion assessment similar to dobutamine stress echocardiography and hence can be utilised to assess viability.

2.1.2.2.3 Direct detection of myocardial necrosis or fibrosis with gadolinium contrast

Both acute and chronic myocardial infarction result in increased extracellular space and gadolinium accumulation leading to a hyper-enhanced appearance in contrast with surrounding normal myocardium. The myocardial involvement is characteristically from subendocardially to epicardially and different in contrast to non-ischaemic cardiomyopathies. LGE-CMR technique is considered the gold standard in vivo imaging modality and has been shown to predict functional recovery prior to revascularisation.[45,46]



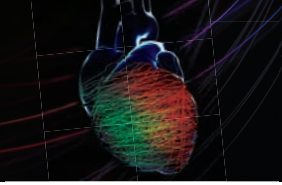
Interestingly presence of unenhanced residual viable myocardial rim of > 3 mm have been shown to be viable despite having myocardial thickness of less than the traditional accepted threshold of 5.5 mm wall thickness for viability.[47,48] A higher scar burden and presence of microvascular obstruction (Figure 3B) have been demonstrated to be associated with higher mortality.[33,49]

2.1.2.3 Assessment in cardiac resynchronisation therapy (CRT)

CRT is a novel device therapy that is indicated in patients with moderate-severe heart failure refractory to optimal medical therapy. While morbidity and mortality are reduced with CRT,[50] up to 30% of patients do not respond to therapy.[51] Though echocardiography is capable of mechanical dyssynchrony assessment, it has been shown to be a poor predictor of response to CRT.[52]

CMR is capable of determining strain and hence dyssynchrony via myocardial tagging sequence. While this technique is still in the realm of research, combined with scar burden assessment, a single centre study has shown promising results with 95% accuracy in predicting functional class improvement (PPV 93%, NPV 100%).[53] Total scar burden has significant correlation to reduction in LV size post CRT.[54] Scar extent and posterolateral location have been shown to be predictors of poor responders.[55] Lastly CMR is also able to identify coronary vein anatomy and guiding lead placement.

While CMR has the potential to provide complete assessment for CRT implantation, utilisation of CMR in the follow-up monitoring is currently limited by the availability of MRI conditional CRT devices and artifacts.



2.1.2.4 Assessment of left ventricular thrombus

Up to 47% of LV thrombi (Figure 1) may be missed by transthoracic echocardiography[56] warranting better detection techniques. Contrast enhanced CMR is superior in comparison to either transthoracic (TTE) or transoesophageal (TOE) echocardiography[57] and is considered the gold standard in non-invasive imaging.

2.1.3 CMR STRESS IMAGING

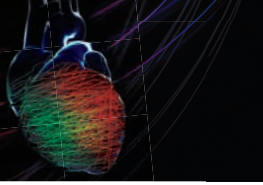
In clinical testing for coronary ischaemia, CMR is done using two distinct methods, wall motion assessment with dobutamine stress CMR or more recently with stress myocardial perfusion CMR.

2.1.3.1 Dobutamine stress CMR

Similar to dobutamine stress echocardiography, dobutamine stress agent is used to induce tachycardia leading to an increase in myocardial O_2 consumption. In presence of inducible ischaemia, regional wall abnormalities can be then detected with the 'cine' sequence in CMR. It has been shown to be safe[58] to perform and has been validated against other modalities.[59]

The ability of CMR to obtain perfect planes across the various long axis and short axis views without angulation errors and window limitations renders it highly reproducible with low interobserver variability.[60] Presence of detected ischaemia has prognostic implications[60] and independently predicts cardiovascular events in patients undergoing non-cardiac surgery.[61]

Dobutamine stress CMR is especially helpful for patients with poor echo windows and have severe renal impairment precluding use of gadolinium contrast. It is best done with a 1.5 Tesla MRI scanner or with 3 Tesla MRI scanner that has parallel or multitransmission technology.



2.1.3.2 Stress perfusion CMR

Stress perfusion CMR, commonly with adenosine is increasingly utilised to detect inducible myocardial ischaemia (Figure 4). Myocardial perfusion can be assessed with CMR by injecting a bolus of gadolinium contrast through a large bore cannula and visualising its passage into the myocardium (first pass perfusion). Comparison is done with second passage of contrast during adenosine administration to look for reversible perfusion defects. This differs from SPECT which use the accumulation of radio – labelled tracer over a longer time in which imaging is performed several minutes after the injection.

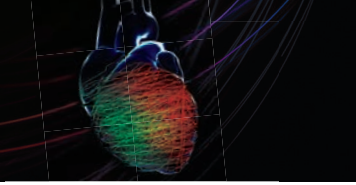
It is superior to exercise treadmill testing,[62] non-inferior and in some aspects superior to SPECT imaging.[5,63] In patients with intermediate lesions on coronary angiography, it is capable of identifying patients at risk despite being on intensified medical treatment.[64] A recent meta-analysis using fractional flow reserve (FFR) as a gold standard showed that adenosine stress perfusion CMR has high sensitivity and specificity (> 0.85) for detecting ischaemia at both patient and coronary artery territory level.[65]

It has to be emphasised that a minimum of 24-48 hour abstinence period from caffeinated food and beverages including tea, chocolate and cola is mandatory prior to testing as they render adenosine inactive and may lead to false negative results.

2.1.3.3 Safety during stress imaging

It is recommended that at least two experienced operators are present; one of whom should be a physician. In addition, the following should be in place:

1. Checklist of contraindications to MRI, contrast and vasoactive drugs



2. Haemodynamic monitoring equipment during scanning (BP, ECG, Pulse oximetry)
3. Emergency patient evacuation procedure out of MRI suite, with regular practice sessions.
4. Emergency resuscitation policy and available equipment i.e. defibrillator and emergency drugs.

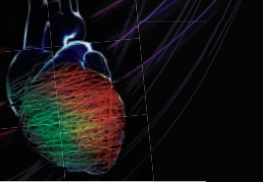
2.2 Non-ischaemic cardiomyopathies

2.2.1 OVERVIEW

CMR has the unique capability of answering important questions in the evaluation of cardiomyopathies. These include quantifying biventricular volume and function, multiple imaging techniques for comprehensive tissue characterisation, infarct/fibrosis imaging with LGE and myocardial perfusion imaging. LGE in particular, provides the key to distinguishing the different types of cardiomyopathies. Non-ischaemic cardiomyopathies usually have patchy, mid-wall, epicardial or global hyperenhancement on LGE imaging, which assists in identifying the aetiology. The presence and degree of LGE distribution also impacts on patients' prognoses as it is also predictive of increased cardiac events despite adjustments for ventricular function and heart failure symptom class. In the following sections, we detail the different cardiomyopathy subtypes and CMR's unique role in diagnosing them.

2.2.2 MYOCARDITIS

Myocarditis is the most common aetiology for patients presenting with acute coronary syndrome but normal coronary arteries.[66] It is most commonly viral in origin and during the first days following infection, there is direct cardiomyocyte



injury followed by oedema, necrosis and finally regional or global contractile dysfunction. In uncomplicated cases, there is usually full tissue and functional recovery within 4 weeks. Severe cases however result in significant myocardial scarring and may result in dilated cardiomyopathy.[67]

The use of echocardiography or nuclear imaging tools has proven to be less-than-useful and CMR has become the diagnostic tool of choice for patients presenting with acute non-ischaemic myocardial injury. CMR allows the examination of several features of myocarditis not available in any other imaging modality.

CMR cine images allow assessment of myocardial volume, function and pericardial effusion. T2-STIR assesses myocardial oedema and LGE assesses inflammatory hyperaemia and necrosis (Figure 5). These features make up the Lake Louise Criteria for the diagnosis of active myocarditis.[68] These criteria and their imaging sequences are tabulated (Table 6) and the presence of any 2 of 3 criteria indicates the presence of active myocarditis. Furthermore, when combined together with perfusion imaging, CMR is able to effectively rule out significant coronary artery disease in the same setting.[69]

Utilisation of the Lake Louise Criteria affords a high specificity and positive predictive value to the CMR evaluation of myocarditis. Moreover, the presence of necrosis or scar has been associated with higher cardiovascular and overall mortality, adding an incremental, prognostic value to CMR evaluation of myocarditis.[70]

In conclusion, CMR is the most comprehensive, accurate, diagnostic and prognostic tool for patients with suspected myocarditis and should be at the forefront for the assessment of acute myocardial injury with normal coronary arteries.

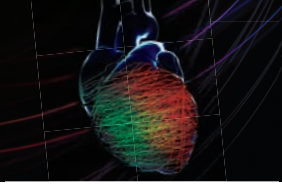
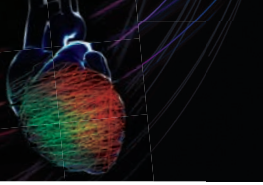


Table 6. Lake Louise Criteria for active myocarditis

Lake Louise Criteria	CMR Technique	CMR Sequence
Presence 2 out of 3 definitive		
Oedema	Oedema imaging	T1-weighted short tau inversion-recovery (STIR)
Hyperaemia	Early gadolinium enhancement	First-pass contrast-enhanced perfusion imaging
Necrosis	Late gadolinium enhancement (LGE)	Phase sensitive inversion recovery (PSIR)
Presence supportive		
Systolic dysfunction	Cine imaging	Steady-state free precession (SSFP)
Pericardial effusion	Cine and/or LGE	SSFP and/or PSIR

2.2.3 FAMILIAL HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disorder; it has an estimated prevalence of 1:500 in the general population.[70] HCM is defined as segmental or diffuse hypertrophy of the left ventricle with a non-dilated and hyperdynamic chamber in the absence of cardiac or systemic disease.[71]



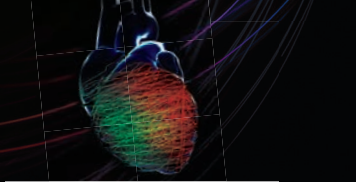
Although there is a range of clinical manifestation for HCM, it is usually the result of systolic and/or diastolic dysfunction, left ventricular outflow tract (LVOT) obstruction, supraventricular/ ventricular arrhythmias and sudden cardiac death (SCD).

The role of imaging in the evaluation of HCM is to diagnose the disease, characterise its phenotype, assess cardiac function (including the presence of dynamic obstruction), to classify disease severity and use as a guide for appropriate therapy, provide risk stratification, and serve as a screening tool for the family.

2.2.3.1 Role of CMR

The diagnosis of HCM traditionally relied on clinical assessment and transthoracic echocardiography (TTE) in identifying unexplained left ventricular hypertrophy (LVH) in the presence of non-dilated left ventricular (LV) cavity. The other supporting features on imaging are systolic anterior motion (SAM) of the mitral valve, and LVOT obstruction. Due to a combination of technical limitations and the highly variable nature of HCM phenotypic expression, TTE assessment is unable to confidently establish or refute a diagnosis of HCM. On TTE the degree of LV hypertrophy could be underestimated which can delay proper treatment, thereby failing to prevent a sudden cardiac death.

LGE is recognised in many disease processes involving the myocardium such as myocardial infarction, myocarditis and cardiomyopathy. LGE has been reported in up to 75% of patients with HCM in whom the vast majority have patchy midwall enhancement which is typically most pronounced within the segments most severely affected by hypertrophy.[72] LGE most often involves the interventricular septum, particularly the anteroseptal mid to basal segments and right ventricular insertion points.[73]



To determine the peak velocity of blood flow through the LV outflow tract in patients with left ventricular outflow tract obstruction (LVOTO), doppler echocardiography is the modality of choice.

CMR is useful in pre-operative planning for surgical myectomy particularly in patients with multilevel LV obstruction and in patients with right ventricular (RV) outflow tract abnormalities. CMR can also quantify the amount of tissue necrosis induced by septal alcohol ablation, as well as the location of scarring and the regression of LV mass following the procedure.

2.2.3.2 Disease characterisation

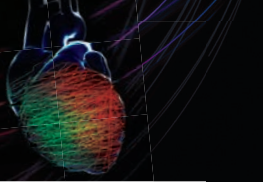
Phenotypic heterogeneity causes great variability in the imaging appearances of HCM.[74] CMR is useful in these variants due to its complete unrestricted coverage of the LV, especially when disease is confined to just a few myocardial segments separated by regions of normal wall.

2.2.3.3 Risk stratification

The criteria for risk stratification of HCM include LV wall thickness, LVOTO, LV dilatation with depressed ejection fraction (burned-out phase), the presence of fibrosis, and perfusion defects.

2.2.3.4 Conclusion

CMR is strongly recommended as a powerful imaging modality for differentiating HCM from other cardiomyopathies, as well as for risk stratification of HCM.



2.2.4 IRON-LOADING CONDITIONS

Iron-loading conditions results in myocardial siderosis and restrictive heart failure. They can occur congenitally such as in hereditary haemochromatosis or acquired such as in ß-thalassemia major. Hereditary haemochromatosis is an autosomal disorder that results in a fractional increase in dietary iron absorption whereas ß-thalassemia major is a recessive disorder that results in ineffective erythropoiesis, profound anaemia and repetitive blood transfusions. Both of these conditions causes saturation of the reticuloendothelial system cells with iron, spilling out to other parenchymal cells and resulting in end-organ damage.[75]

CMR allows measurement of right (RV) and left (LV) ventricular volumes and systolic function, and accurate LV mass calculation. LGE detects myocardial replacement fibrosis which is known to occur in iron-loaded, restrictive cardiomyopathy.[76] More importantly, CMR offers the ability to reproducibly quantify myocardial iron deposition without the need to resort to biopsy.

Myocardial T2* which is a relaxation parameter that is increased with iron deposition. Its quantification can be used not only to diagnose iron loading in susceptible individuals, it can also be used to monitor treatment as an improvement in myocardial T2* has been shown to be associated with an improvement in the LV ejection fraction (LVEF).[77]

The lower limit of normal for T2* measurement in healthy non-anaemic subjects is 20 ms and this equates to 1.1 mg/g iron dry weight.[78] Ninety-eight percent of patients with congestive cardiac failure (CCF) symptoms had T2* < 10ms. A 3-tiered risk model has since been developed based on this finding and this is illustrated in Table 7. A normal myocardial T2* has a high predictive value for exclusion of CCF for 12 months.[79]

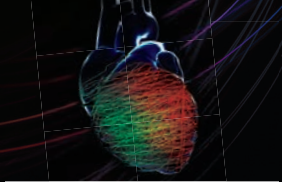


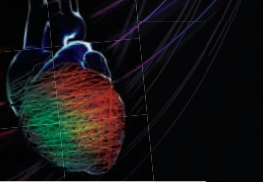
Table 7. 3-tiered risk model

T2* (ms)	Risk of CCF within 12 months
> 20	Low
10-20	Intermediate
< 10	High

2.2.5 AMYLOIDOSIS

Amyloidosis is classified into many different forms on the basis of the amyloid precursor protein. Cardiac accumulation of these various proteins in the insoluble fibrillar amyloid conformation occurs principally in the myocardial interstitium.[80] These often is associated with endomyocardial fibrosis leading to diastolic dysfunction.

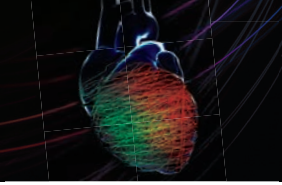
Various CMR sequences have been shown to be useful in assessing cardiac amyloidosis. A diffuse decrease in signal intensity on T1-weighted fast spin echo images may be found in cardiac amyloid.[81-83] There is also usually generalised myocardial thickening affecting the right and left ventricles in a diffuse pattern rather than a focal hypertrophy. Thickening (> 6 mm) of the interatrial septum and posterior right atrial wall is also suggestive of cardiac amyloidosis.[84] Marked thickening of the LV wall is associated with a survival time of less than 6 months.[85] Late-enhancement sequences have been found to demonstrate diffuse (Figure 11) or subendocardial late enhancement pattern.[86,87] It can be challenging to depict these areas on cardiac MR images, because the total amyloid protein load results in rapid washout of gadolinium-based contrast material from the myocardium.[2] Thus, it can be difficult to determine the optimal



inversion time to null normal myocardium (Figure 10), because it may be unclear which myocardial areas are normal.[88] Focal enhancement correlates significantly with areas of regional hypokinesis or akinesis.[87]

When compared with the diagnostic test of choice of endomyocardial biopsy, cardiac MR imaging provides good sensitivity (80%) and high specificity (94%).[88] One study showed that the abnormal gadolinium kinetics, specifically the 2-minute post gadolinium intramyocardial inversion time difference between subepicardium and subendocardium at a threshold value of 23 msec, predicted mortality with 85% accuracy.[89] A study by Maceira *et al.* of 29 patients with systemic amyloidosis and 16 hypertensive controls showed that amyloidosis was associated with qualitative global and subendocardial CMR gadolinium enhancement of the myocardium.[86] Global subendocardial LGE was found in approximately two-thirds of patients.[86] Based on pathological correlates in a patient from this study, the CMR hyper-enhancement probably represents interstitial expansion from amyloid infiltration.

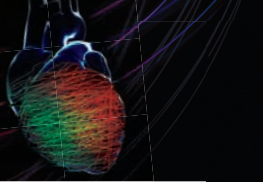
In another study by Perugini *et al.* using an Italian population of patients with histologically proven systemic amyloidosis and echocardiographic diagnosis of cardiac involvement, gadolinium enhancement by CMR was detected (Figure 11) in 16 of 21 (76%) patients.[90] In contrast to the study of Maciera *et al.*, where the pattern of late enhancement was global and subendocardial, Perugini *et al.* reported a much more variable pattern of late enhancement, that could be localised or diffuse, and subendocardial or transmural.[90, 91] Syed *et al.* performed LGE CMR in 120 patients referred to a tertiary centre with confirmed amyloidosis; 97% of the histologically confirmed cardiac amyloidosis (35/120) had abnormal LGE and 91% had increased LV wall thickness on echocardiography.[91, 92] Eighty-three percent had global LGE pattern (transmural and subendocardial).[92]



2.2.6 SARCOIDOSIS

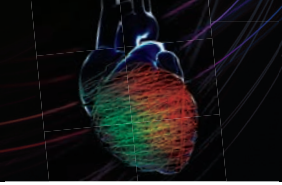
Sarcoidosis is a granulomatous disease of unknown aetiology that can affect any organ. Cardiac involvement is uncommon but it has a wide spectrum of clinical manifestations and is potentially fatal.[93] Cardiac abnormalities are caused by infiltration of sarcoid granulomas.[94] The Japanese Ministry of Health and Welfare (JMHW) guidelines for diagnosis of cardiac sarcoid, which incorporates the use of ECG, cardiac imaging and histopathology, is the most well-known and is currently the standard.[91,95] Mehta *et al.* found that including advanced cardiac imaging with positron emission tomography scanning or CMR increased sensitivity above the previously established criteria.[91,96]

CMR is particularly useful in identifying even small areas of myocardial oedema and fibrosis leading to post-inflammatory scarring that is typically seen in cardiac sarcoidosis.[91] Both global and regional contractile dysfunction has been described, although, similar to cardiac amyloidosis, the LGE technique has been most widely evaluated in clinical studies using CMR (Figure 12). The LGE patterns are characteristically patchy and usually appear in the midwall and subepicardium but may involve any layer of ventricular myocardium and can mimic coronary artery disease distribution.[81,97] The RV myocardium and RVOT may also show enhancement. If a tissue diagnosis is required, cardiac MR imaging may provide optimal guidance for endomyocardial biopsy.[81,98] Smedma and colleagues evaluated the utility of LGE in 58 patients with biopsy proven pulmonary sarcoidosis, 25% of whom also had symptoms suggestive of cardiac involvement.[91,99] CMR revealed LGE, mostly involving the epicardium of the basal and lateral segments, in 73% of patients diagnosed with cardiac involvement by the Japanese criteria.[91,99] Other studies have confirmed the predilection of



LGE for the basal–lateral segments, although subendocardial or transmural hyperenhancement has been also observed, mimicking the ischaemic pattern.[91] Recently, Patel and colleagues prospectively studied 81 patients with biopsy proven extracardiac sarcoidosis to compare between LGE CMR and standard clinical evaluation.[100] In their study, LGE CMR identified myocardial abnormalities in significantly more patients than a standard clinical evaluation based on JMHW guidelines (26% vs 12%). LGE CMR positive patients had a higher rate of adverse events, including cardiac death, during a 21 month follow-up, compared to LGE CMR negative patients.[100]

Myocardial damage detected by LGE CMR appears to be associated with future adverse events including cardiac death, but there were few events in this small cohort and therefore a large scale study is required. Apart from LGE, both functional and anatomical ('white blood' and 'black blood') CMR sequences can help in detecting cardiac sarcoid by demonstrating some of its characteristic features—septal thinning, biventricular dilatation/systolic dysfunction, and pericardial effusion.[91] T2 weighted sequences may also help in identifying myocardial oedema, and may also depict sarcoid lesions as patchy hyperintense areas in myocardium.[101] CMR also identifies pulmonary features of sarcoid, such as enlarged hilar lymph nodes and lung fibrosis (Figure 12).[91] Improvements in cardiac symptoms and MR findings have been observed after steroid therapy.[101, 102]

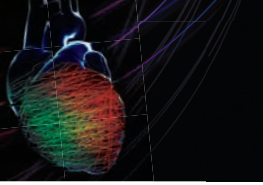


2.2.7 ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY/DYSPLASIA

Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) is a genetically inherited desmosomal protein disorder characterised by progressive fibrofatty replacement of the right ventricular myocardium.[103] In majority of patients, it is inherited as an autosomal dominant with incomplete penetrance but in a small group of population, it is inherited as an autosomal recessive form, called "Naxos disease", of which in addition to ARVC/D, there are skin (palmoplantar keratoses) and hair (woolly hair) manifestations.[104,105] Its prevalence in Asian population remains unknown, however, ethnic difference in the occurrence of the disease exists. An Italian study gives estimated prevalences for ARVD/C ranging from 1 in 2,000 to 1 in 5,000 whilst in contrast, a Japanese study has reported a prevalence of only 1 in 160,000.[106,107]

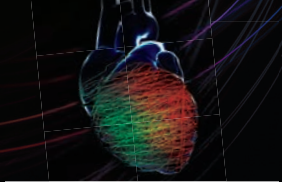
The original 1994 International Task Force criteria for the clinical diagnosis of ARVC/D has been revised to incorporate new emerging diagnostic modalities to improve diagnosis. The modified Task Force criteria for ARVC/D diagnosis 2010 incorporated CMR as one of the imaging technique of choice in the assessment of ARVC.

Imaging modalities commonly used for ARVC/D evaluation include echocardiography, CMR, and RV angiography. Both echocardiography and angiography have significant limitations in assessing the RV due to its complex geometry. Over the last decade, CMR has emerged as the imaging modality of choice in ARVC/D, allowing for non-invasive morphological and functional evaluation, as well as tissue characterisation in a single investigation.



Major criteria
Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following: • Ratio of RV end-diastolic volume to BSA > 110 mL/m^2 (male) or > 100 mL/m^2 (female), or • RV ejection fraction < 40%
Minor criteria
Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following: • Ratio of RV end-diastolic volume to BSA > 100 to < 110 mL/m^2 (male) or > 90 to < 100 mL/m^2 (female), or • RV ejection fraction > 40% to < 45%

CMR can detect fatty infiltration in the right ventricle and occasionally in the left ventricle however, fat infiltration of the ventricles is currently only a major criterion for diagnosis of ARVC on cardiac biopsy.[108]

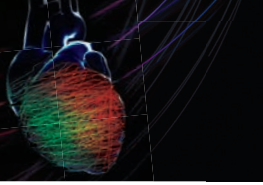


2.2.8 LEFT VENTRICULAR NON-COMPACTION

Left ventricular non-compaction (LVNC) is a rare cardiomyopathy associated with an autosomal dominant inheritance and characterised by the presence of an extensive non-compacted myocardial layer lining the cavity of the left ventricle.[109] Prominent trabeculation and deep intertrabecular recesses is characteristically seen, alters myocardial structure and could potentially lead to cardiac failure, thromboembolism and malignant arrhythmias. There is no treatment for LVNC but early diagnosis allows institution of standard heart failure and anticoagulation therapy which prevents complications.[110]

CMR provides a high spatial resolution, lower investigator dependency, no viewing limitations in any tomographic plane and it is not limited by a patient's constitution. This is important as it allows imaging of the entire volume of the heart which is important as most trabeculations are found in the mid to distal, anterior, lateral and apical segments.[109]

A superior contrast between trabeculation and blood pool also allows more precise calculation of the non-compacted to compacted (NC/C) ratio calculation with a NC/C ratio of > 2.3 in diastole being pathognomonic for pathological non-compaction, with a high degree of sensitivity (up to 100%), specificity (93%), positive, and negative predictive certainty (up to 100%) compared to echocardiogram (Figures 13A and 13B).[111] Additionally, incremental information, such as presence of a left ventricular thrombus (Figure 1) and myocardial fibrosis, can be readily acquired with LGE which has also been shown to have a potentially prognostic factor in LVNC patients (Figures 13C and 13D).[112]

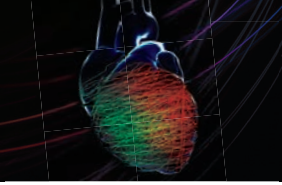


2.2.9 *TAKO-TSUBO* CARDIOMYOPATHY

Tako-tsubo is a stress-induced cardiomyopathy initially reported in Japan, characterised by acute left ventricular dysfunction with relative basal-sparing and typically described as 'apical ballooning'.[113] Evidence suggests that enhanced sympathetic activity is the pathological basis behind the transient myocardial dysfunction. A majority of patients present as acute coronary syndrome but with normal or non-obstructive coronary arteries.

CMR is helpful in distinguishing between reversible injury (inflammatory or ischaemic oedema) and irreversible injury (necrosis or fibrosis) which is important for excluding pathologies that may present with similar symptoms, such as myocarditis or myocardial infarction.[114] Additional incremental information is also made available such as presence of pleural or pericardial effusion and ventricular thrombi.

The presence of LGE in the acute phase has also been suggested to be associated with worse symptoms (cardiogenic shock more prevalent) and prolonged recovery of clinical findings (longer recovery for ECG and wall motion abnormalities). Therefore CMR provides excellent diagnostic utility, additional incremental information to effectively rule out differentials and prognostic information.[115]



2.2.10 ANDERSON-FABRY DISEASE

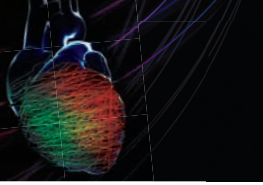
Anderson–Fabry disease is an X-linked recessive lysosomal storage disorder that is caused by a deficiency of the lysosomal enzyme ß-Galactosidase A (also termed ceramide trihexosidase).[116] Cardiac involvement is very common and represents the most important cause of death.[117] The unremitting accumulation of glycosphingolipids in cardiac structures can lead to a variety of cardiac signs and symptoms, including LV hypertrophy, arrhythmias, coronary artery disease (mainly small-vessel disease) and heart failure.[116]

CMR is the technique of choice for a suspected Anderson-Fabry disease as it frequently enables a specific diagnosis to be established. It is important to distinguish Fabry disease from other causes of left ventricular hypertrophy (LVH) including hypertensive heart disease and amyloidosis.

In most cases the LVH is concentric; however, an asymmetrical variety with septal thickening and posterior wall fibrotic thinning may present in severe cases. Right ventricular hypertrophy is also common and may progress to right ventricular dilation.

On LGE, hyperenhancement is typically seen midwall in the basal inferolateral segment with sparring of the endocardium. LGE may also have a prognostic role with a recent study suggesting its presence predicts a lack of response to enzyme replacement therapy, presumably due to irreversible myocardial tissue damage.[118]

Anderson-Fabry disease should be considered in the differential diagnosis of patients with unexplained myocardial hypertrophy of the left ventricle, or hypertrophic or restrictive cardiomyopathy. There are also cases that gradually develop dilated cardiomyopathy.



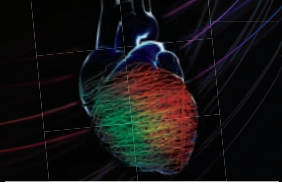
2.2.11 PERIPARTUM CARDIOMYOPATHY

Peripartum cardiomyopathy (PPCM) is an uncommon form of cardiomyopathy afflicting pregnant ladies around the time of delivery. Malaysia has a similar case rate as the United States, Canada, and Europe, with an estimated 1 per 2500 to 4000 live births.[119,120] It is commonly associated with older maternal age, multiparity, multifoetal pregnancy (e.g. twins), hypertension, prior toxin exposure (e.g. cocaine) or use of certain medications to prevent premature labour. The aetiology is not fully understood and several physiopathological mechanisms have been proposed, including viral myocarditis, abnormal immune response or haemodynamic stress during pregnancy and increased myocytes as well as cytokine-mediated activity. Owing to the complexity of the physiopathology, the prognosis remains difficult to predict. Outcome is variable with 50% of patients with PPCM recover normal heart function, 25% have persistently reduced heart function but remain stable on medications, and 25% progress to severe heart failure.[120]

PPCM is diagnosed when the following three conventional criteria are met:[121]

1. Heart failure develops in the last month of pregnancy or within 5 months of delivery.
2. Reduced systolic ejection fraction (EF) less than 45% (typically measured by an echocardiogram).
3. No other cause for heart failure with reduced EF can be found.

CMR is not generally required to make the diagnosis of PPCM but it can be helpful in demonstrating LV/RV dilatation and systolic dysfunction particularly if echocardiography is suboptimal.

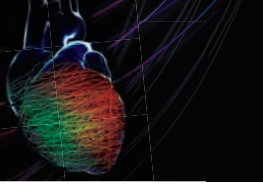


Case reports and small series have noted variable presence of LGE in patients with PPCM. This variability likely reflects the diverse processes that lead to PPCM. Focal non-ischaemic LGE and regional wall motion abnormalities were evident predominantly located in the anteroseptal and basal to midventricular segments.[122] The presence and persistence of LGE may be associated with poor recovery of cardiac function; improving LGE may be associated with cardiac recovery, while lack of LGE may be associated with presence or absence of cardiac recovery.[123-125] However, the prognostic value of CMR in PPCM has not been established.

Safety studies have been performed predominantly at or below 1.5 Tesla magnetic field strengths and showed no reported harmful effects from CMR of the pregnant woman or foetus. There may be an increased risk of tissue heating at higher field strengths. Gadolinium crosses the placenta and is excreted by the foetus into the amniotic fluid. It is then swallowed so it can be reabsorbed into the foetal circulation. Given the potentially long half-life in the foetus and few data from human pregnancy, the risk of gadolinium should be justified against benefit. Breastfeeding can be continued without interruption after the use of gadolinium.

2.2.12 DILATED CARDIOMYOPATHY AND ETHANOL-INDUCED CARDIOMYOPATHY

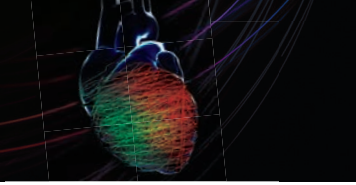
Dilated cardiomyopathy (DCM) is defined as the presence of a left or biventricular dilatation with impaired systolic function in the absence of abnormal loading conditions such as hypertension, structural heart disease, valvular heart disease or ischaemic heart disease, sufficient to cause global systolic impairment.[126] The aetiology is also heterogeneous. Among toxic forms, alcoholic cardiomyopathy is the most frequent aetiology of secondary DCM and accounts for approximately 4% of all cardiomyopathies.[127]



Ethanol-induced cardiomyopathy (ELC) is a type of acquired dilated cardiomyopathy associated with long-term heavy alcohol consumption and has been implicated in as many as one third of cases of dilated cardiomyopathy. The mechanism by which ethanol causes cardiac damage remains unclear. Several theories have arose based on human and animal studies including oxidative stress, apoptosis, mitochondrial dysfunction, derangements of fatty acid metabolism/transport and accelerated protein catabolism.[128]

CMR has become the gold standard method for the assessment of cardiac morphology and function in various cardiomyopathies. About 60% of patients with DCM showed no detectable fibrosis. In 30% of cases, LGE has been described with the most common pattern being characterised by a midwall linear distribution likely representing the intramural layer of septal fibrosis which has been observed in pathologic samples.[129]

Findings in ELC is similar to cardiomyopathy of non-ischaemic aetiology such as globally decreased in decreased ventricular function, chamber dilatation, mitral and tricuspid incompetence from annular dilatation and evidence of pulmonary hypertension such as dilated pulmonary arteries. It has been traditionally associated with mid-wall enhancement during LGE study. However, there has been an emerging evidence of myocardial involvement in patients with ELC resembling appearance of myocarditis.[130] The patchy pattern of LGE may not be due to fibrosis, but rather caused by pathological deposits (e.g. accumulated cardiotoxic metabolites, oxygen supply/demand mismatch due to arteriovenous pulmonary shunts, inflammation, a yet unknown mechanism). This might be explained by the findings of previous studies, which showed that cardiomyocytes and the trabecular network of the myocardium are changed in patients with liver cirrhosis.[131]



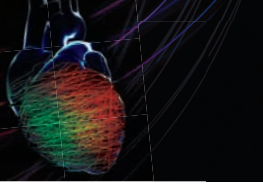
When the identified cause is treated, some may exhibit recovery of the cardiac function and CMR is ideal for interval imaging for quantification due to its accuracy, reproducibility and lack of radiation. The natural history of patients with ELC depends greatly on a patient's ability to cease ethanol consumption completely. Multiple case reports and small retrospective and prospective studies have clearly documented marked improvement or, in some patients, normalisation of cardiac function with abstinence. Recognition of alcohol as a potential cause of cardiomyopathy is crucial since abstinence can result in an improved ejection fraction in 50% of the patients medically treated for heart failure, and continued drinking can result in further deterioration of the cardiac function.[127]

2.3 Congenital heart disease

CMR is an excellent diagnostic and assessment tool for patients with congenital heart disease, both children and adult alike. It gives structural and haemodynamic data, the latter in the form of flow rather than pressure. In many centres, it has superseded cardiac catheterisation especially in larger children and adults with congenital heart disease as the image quality is superior to that of transcatheter angiograms. The downside however, is the need to hold breath during the scan, a task which can be overcome by endotracheal intubation and general anaesthesia. Common indications for congenital CMR scans are as follows:

2.3.1 TETRALOGY OF FALLOT

CMR has evolved as the reference standard imaging modality in both pre and postoperative assessment of patients with Tetralogy of Fallot (TOF).



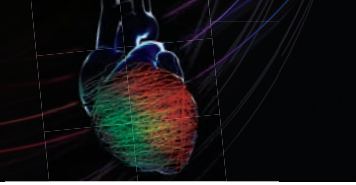
Preoperatively it defines the level of right ventricular outflow obstruction, the calibre of pulmonary valve and branch pulmonary arteries. It also evaluates and quantifies the aorto-pulmonary collateral flows, which may cause left sided heart failure immediately post-surgery.

Following surgical repair, longstanding pulmonary regurgitation may lead to progressive RV dilatation, dysfunction and sudden death. The strength of CMR is in its ability to perform serial, interrogation of the right ventricle and pulmonary regurgitation. This enables determination of optimal timing of pulmonary valve replacement. Recent studies have also shown an association of myocardial fibrosis with increased risk of sudden death.

In patients who had a Rastelli type (VSD closure and right ventricle to pulmonary artery conduit placement) of repair in a subset of TOF, CMR is often used to interrogate conduit dysfunction, mode and timing of intervention. This is additional to routine right ventricular assessment as described above.

2.3.2 ATRIAL SEPTAL DEFECT

Transoesophageal echocardiography is the most widely used imaging modality to assess the morphology of atrial septal defects (ASD) and to guide transcatheter device placement. CMR may serve as an adjunctive tool in providing haemodynamic data such as shunt magnitude (Qp:Qs ratio), right ventricular volume and function to guide clinical management. Qp:Qs ratio compares flow between the main pulmonary artery and the aorta and has good correlation with values obtained by invasive catheterisation.[132] Direct en face view of the ASD allows visualisation of the size, shape and location of the ASD.[133] Any associated anomalies such as partial anomalous pulmonary venous drainage can also be demonstrated.



2.3.3 COARCTATION OF AORTA

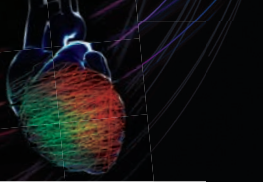
CMR allows complete evaluation of the entire aortic arch including severity of coarctation, the morphology of the adjacent arch segments such as transverse arch hypoplasia and associated lesions such as bicuspid aortic valve. It provides 3D visualisation of arch geometry to enable clinical decision-making such as selection of treatment modalities (balloon angioplasty, stent implantation or surgical repair) and detection of post-interventional complications (re-stenosis, aneurysm formation, dissection).

Measuring the peak velocity across the coarctation allows estimation of its severity. Collateral flow can also be assessed by comparing flow pre-stenosis with the flow at the level of the diaphragm.[134]

2.3.4 TRANSPOSITION OF GREAT ARTERIES

Patients with transposition of great arteries (TGA) were previously repaired by atrial switch operation (Mustard or Senning). These patients are at risk of complications such as atrial arrhythmias, baffle obstructions and failing systemic right ventricle. CMR comes in as a monitoring tool to assess the patency of atrial baffles as well as for baffle leak. More importantly, it is to assess the true systemic right ventricular function, which is often masked by tricuspid regurgitation.

In the modern era, arterial switch is the operation of choice due to better long term outcome. These patients may develop pulmonary artery obstruction following Le-Compte maneuvre or neo-aortic regurgitation; both of which are best assessed with CMR. Due to coronary artery re-implantation during initial surgery, patients with single coronary origin are at higher risk of coronary ischaemia and therefore CMR adenosine perfusion testing may be attempted.



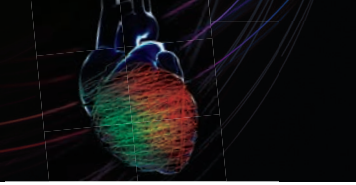
2.3.5 CONGENITALLY CORRECTED TRANSPOSITION OF GREAT ARTERIES

Periodic CMR is helpful in determining the timing of repair, which largely depends on the ability of RV to maintain a balanced, systemic circulation. The assessment of right ventricle as the systemic ventricle is best made with CMR especially the presence of varying degree of tricuspid regurgitation (left atrio-ventricular valve). Additionally, CMR provides excellent spatial resolution to both the cardiologists and surgeons to draw out surgical plan e.g. how best to place the conduit, the baffle and left ventricular outflow tunneling.

Following surgical repair, CMR is essential in the follow up assessment of this group of patients who are either repaired physiologically or anatomically.

2.3.6 UNIVENTRICULAR HEART

Patients with functional single ventricle congenital heart diseases are commonly palliated with Fontan procedure. CMR allows assessment of the entire Fontan pathway. This includes the cavo-pulmonary anastomotic sites, branch pulmonary arteries, pulmonary veins, the atrio-ventricular valves, ventricular outflow tracts and aortic arch for detection of residual stenosis, leaks or collaterals and intraatrial thrombus.[135] It is the best modality for the assessment of ventricular size and function of the functional single ventricle. In combination with detection of myocardial fibrosis, it predicts those who are at risk of failing Fontan and poor outcome.[136]



2.3.7 EBSTEIN ANOMALY

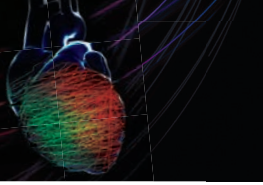
Ebstein anomaly is characterised by apical displacement of the septal and posterior leaflets of the tricuspid valve, resulting in atrialization of part of the right ventricle. Tricuspid regurgitation is common and progressive RV dilatation and dysfunction eventually leads to biventricular failure.

The morphology of the tricuspid valve leaflets as well as the severity of the regurgitation is better visualised compared to echocardiogram.[137] Volumetric quantification of the functional RV and atrialised RV also forms part of preoperative evaluation and CMR provide more accurate measurements with good reproducibility.[138]

2.4 CMR in valve assessment

CMR has a velocity encoding or phase contrast sequence that renders it capable of quantifying blood flow accurately.[139] This includes quantifying flow across great vessels and as such the net RV/LV cardiac output can be determined and the blood flow across the aortic and pulmonary valves can be measured to determine if there is stenosis or regurgitation based on similar method in echocardiography. CMR assessment of the various valvular lesions has been validated against echocardiography[140-143] and is consistently reproducible.[144]

Whilst echocardiography remains the first line and modality of choice due to its ubiquity, higher temporal resolution and ability to view thin and mobile structures better,[145] CMR can be a complementary modality when echocardiography becomes challenging in patients with poor echo window and is especially useful in right sided heart valve and morphology assessment.



2.4.1 ESTIMATION OF SEVERITY OF VALVULAR LESIONS

Flow related signal loss (dephasing) in the presence of valvular stenosis or regurgitation in the cine sequence gives a preliminary overview of the existence of a valvular lesion just as a Colour Doppler would demonstrate in echocardiography.[146,147]

2.4.1.1 Valvular stenosis

Further quantification of stenotic valve lesions is done by prescribing an imaging plane directly across the valve of interest and calculated using:

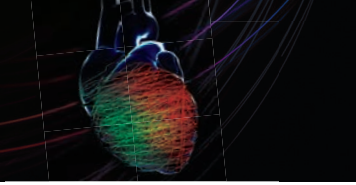
a) Planimetry using cine sequence
b) Measurement of the peak velocity and hence pressure gradient using the phase contrast sequence.

Quantification of aortic stenosis by pressure gradient and peak velocity method using the phase contrast sequence has been validated against transthoracic echocardiography[148,149] while the planimetry method has been validated against transoesophageal echocardiography.[150-151] In mitral stenosis similar validation has been done comparing it with transthoracic echocardiography and direct invasive measurement during cardiac catheterisation.[148,152-154]

2.4.1.2 Valvular regurgitation

Regurgitation volume and hence regurgitation volume, on the other hand is calculated based on the either:

a) Directly by placement of the imaging plane across the valve and quantifying backward flow or
b) Indirectly by comparing and calculating from RV and LV stroke volumes, aortic and pulmonary forward and backward flow to derive the regurgitation volume across the atrio-ventricular (AV) valve.

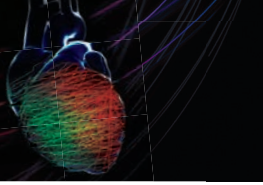


In regurgitant lesions, quantification of regurgitant volume is well validated against echocardiographic and cardiac catheterisation measurements and in relation to pulmonary regurgitation remains a superior method for assessment.[145]

Lastly with the ability to perform tissue characterisation, CMR is helpful in assessing and characterising certain valvular masses provided that the mass is large enough.[155,156] The exception is tissue calcification which is inherently CMR's weakness.

Table 8. Advantages and disadvantages of valve assessment using CMR

Advantages	Disadvantages
No restriction from poor window.	Poor visualisation of thin, mobile structures.
No restriction of imaging plane – assess right sided valve lesions well, particularly pulmonary valve.	Multivalvular lesions reduces accuracy of severity assessment. Slightly underestimates stenotic lesions.
Quantifies flow and regurgitation across relatively immobile and tubular aorta and pulmonary artery - useful in aortic and pulmonary valve lesions.	Quantifies flow and regurgitation less accurately across highly mobile planes e.g. mitral and tricuspid valves.
Limited tissue characterisation of valve masses possible.	Unable to detect calcium well.



2.5 MR angiography and MR coronary angiography

CT scan is the preferred choice for angiography due to shorter scan duration. However, MR angiography is able to provide additional information (e.g. functional assessment) and involves no radiation. This is particularly useful for congenital cases (e.g. coarctation of aorta) that usually require serial monitoring.

2.5.1 INDICATIONS OF MR ANGIOGRAPHY (MRA)

2.5.1.1 Thoracic aorta

Indications:

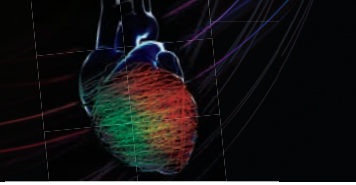
1. Defining the location and extent of aortic aneurysms, erosions, ulcers and dissections.
2. Evaluating post-surgical disease processes and complications involving the aorta and surrounding structures.

The 3D Contrast -enhanced MRA (CE-MRA) technique may be combined with other techniques to provide morphological and functional information that CT is not able to provide.

2.5.1.2 Peripheral arterial disease (PAD)

Indications:

1. To diagnose anatomic location and degree of stenosis in patients with claudication.
2. To aid in patient selection for lower extremities PAD intervention.



The image acquisition needs to include from at least the aortic bifurcation through the distal trifurcation vessels (and pedal arch for limb-threatening ischaemia). CE-MRA is more sensitive and specific than colour duplex ultrasound.[157] When compared to CT angiography as the initial modality of choice there is no statistical difference in patient outcomes.[158]

2.5.1.3 Renal arterial disease

MRA allows for assessment of intra-abdominal arterial stenosis, with recent studies utilising 3.0-T MRA demonstrating high sensitivity (100%) and specificity (92%).[159,160]

Irregularity, dissection, or aneurysmal dilatation of renal arteries can also be identified by this method.

Combining CE-MRA with phase contrast flow quantification gives the advantage of performing both morphological assessment and complementary flow-related data quantification of individual renal arteries.

2.5.1.4 Carotid arteries and intracranial arterial disease

MRA has an established role in carotid artery disease as well as intra and extra cranial arteries diseases. CE-MRA demands machine hardware to generate sufficiently high spatial resolution during the first pass of a contrast agent.

2.5.2 INDICATIONS OF MR CORONARY ANGIOGRAPHY

Currently CT scan is considered the foremost non-invasive alternative to coronary angiography to detect and rule out coronary artery disease. However, CMR is an attractive option, particularly for the proximal part of the coronary arteries.

SECTION 2: INDICATIONS

Indications of MR coronary angiography:

1. Identify coronary artery anomalies.
2. Identify coronary artery aneurysms.
3. May be useful to determine coronary artery patency.

MR coronary angiography is technically more challenging than CMR of other vascular beds due to several unique issues. Among them:

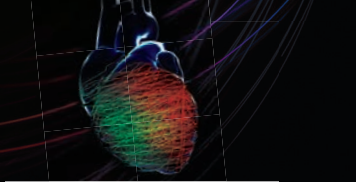
1. Small calibre of the coronary arteries (3-6 mm diameter).
2. Near constant motion of coronary arteries (during both respiratory and cardiac cycles).
3. High level of tortuosity of coronary arteries.
4. Surrounding signal from adjacent epicardial fat and myocardium.
5. Coronary stents may affect image quality as it causes local signal void/image distortion.

To overcome these obstacles, various methods and techniques are employed e.g. cardiac triggering, respiratory motion suppression and 3D image acquisition.

Bright blood (segmented k-space GRE and SSFP) is typically used without any exogenous contrast agent. The free-breathing, navigator-gated 3D- segmented GRE method is the currently the commonest technique used.

CMR is highly advantageous for identifying aneurysms or fistula without the use of contrast materials or exposing patients to ionising radiation. This is particularly important in children and relatively young women.

Data regarding the clinical utility of MR coronary angiography for coronary artery stenoses are based on high-risk populations referred for X-ray angiography. No data for regarding the use of MR coronary angiography for patients presenting with chest



pain or for screening purposes. MR coronary angiography may be considered as part of a complete CMR assessment for patients with dilated cardiomyopathy in the absence of clinical infarction.

MR coronary angiography is an evolving field with other methods constantly been developed. Among them are whole-heart SSFP coronary CMR method. Although it utilises an inferior in-plane spatial resolution, data appear to be at least as accurate as free-breathing methods.

2.6 CMR in cardiac masses and pericardial disease

Cardiac tumours are rare with an estimated prevalence of 0.03% for primary tumours and up to 2% in all tumours.[161] Symptoms and prognosis of cardiac tumours depend on size, location and intrinsic aggressiveness. Significant morbidity of cardiac masses are related to obstruction, infiltration, thromboembolism, arrhythmias or death. Cardiac masses are often identified on other imaging modalities and are referred to cardiovascular magnetic resonance (CMR) for better characterisation.

Echocardiography is generally employed as first line imaging modality as it is non-invasive, cost effective, quick and widely available. CMR has superior tissue contrast resolution and offers multiplanar anatomical evaluation in terms of size, extents and anatomical relationship of masses to neighbouring structures, thus paracardiac, metastatic and infiltrative processes can be confidently evaluated. Cardiac functional assessment and tissue characterisation are often evaluated during the study. The use of contrast agents will enhance detection and delineation of the masses.[162]

The most frequent cardiac mass is the intracavitary thrombus[163] (Figure 1) presented as filling defect in the atrias and ventricles. Secondary deposits are more frequent than primary cardiac

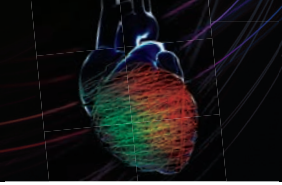
tumours. The metastases may spread through local infiltration which include carcinoma of lung or breast, haematogenous spread as seen with malignant melanomas, leukaemias and lymphomas or, transvenous spread which include renal cell carcinoma or hepatoma. The majority of primary cardiac tumours are benign with most frequent encountered are atrial myxomas (Figure 21) and cardiac lipomas. Malignant primary cardiac tumours are frequently sarcomas.

A number of different imaging sequences are employed for a comprehensive CMR assessment. Black-blood prepared sequences are mainly used for tissue characterisation and assessment of lesion enhancement characteristics following perfusion imaging. Bright blood prepared sequences provide functional information, such as lesion mobility and impact on adjacent valves and chambers.

The pericardium is a thin double-layered sac that holds the heart within the anterior mediastinum. The pericardial cavity normally contains between 10 to 50 ml of fluid.[164] It is normally seen in CMR as a thin, smooth, low-intensity curvilinear structure surrounded by high-intensity mediastinal and epicardial fat, or medium-intensity myocardium. On CMR, normal pericardium measures 1.2 mm and 1.7 mm in diastole and systole respectively. During CMR, the pericardium is assessed morphologically from its thickness and signal homogeneity, abnormal amount and distribution of fluid or fat and, any late contrast enhancement. It is equally important to determine the impact of abnormal pericardium on cardiac filling.

The congenital causes of pericardial anomalies include pericardial cyst (Figure 24), pericardial defect and pericardial diverticulum. The acquired pericardial diseases include pericardial effusion which may be seen in heart failure, renal insufficiency, infection, neoplasm, trauma and myocardial infarction. Inflammatory pericarditis, constrictive pericarditis and pericardial masses are other important causes of acquired pericardial diseases.

CMR may not always provide a definitive pathological diagnosis but it can aid in limiting differential considerations and be of help in treatment planning for surgical cases.



2.7 Extra cardiac findings in CMR

In addition to evaluating the heart and great vessels, the field of view (FOV) of CMR includes the thorax and upper abdomen. CMR scans can therefore detect findings that are incidental to the initial indication that an examination was requested. Extra-cardiac findings in clinical CMR are common in patients referred to CMR (26%).[165] Radiologists and cardiologists have to be aware of relevant extra-cardiac findings which might require additional diagnostics or treatment. It is recommended to have co-reporting by radiologists and cardiologists to ensure a comprehensive evaluation. Studies have shown that 99% of all extra-cardiac findings are detectable on either SSFP scout or black-blood fast spin echo sequences (e.g. HASTE).[166]

Table 9. Examples of significant findings in CMR which require further clinical correlation or investigation.

Significant finding	
Vessels	Congenital and acquired vascular abnormalities
Chest and mediastinum	Pulmonary infections / mass / nodule Pleural effusion / mass Mediastinal lymphadenopathy Oesophageal abnormalities Large airway abnormalities
Upper abdomen	Organomegaly Mass Free fluid
Bones	Infection Mass Deformities
Breast pathology	Mass

References & Acknowledgements

REFERENCES

1. Pohost GM. The history of cardiovascular magnetic resonance. *JACC Cardiovasc Imaging.* 2008;1(5):672-8.
2. NHS England. 2013/14 NHS Standard Contract for Cardiology: Cardiac Magnetic Resonance Imaging (CMR) (Adult). 2013. Available at http://www.england.nhs.uk/wp-content/uploads/2013/06/a09-cari-cardiac-mag-reson-imaging.pdf.
3. Kramer CM. Integrated approach to ischemic heart disease. The one-stop shop. *Cardiol Clin.* 1998;16(2):267-76.
4. Ricciardi MJ, Wu E, Davidson CJ, Choi KM, Klocke FJ, Bonow RO, *et al.* Visualization of discrete microinfarction after percutaneous coronary intervention associated with mild creatine kinase-MB elevation. *Circulation.* 2001;103(23):2780-3.
5. Greenwood JP, Maredia N, Younger JF, Brown JM, Nixon J, Everett CC, *et al.* Cardiovascular magnetic resonance and single-photon emission computed tomography for diagnosis of coronary heart disease (CE-MARC): a prospective trial. *Lancet.* 2012;379(9814):453-60.
6. Salerno M, Kramer CM. Advances in parametric mapping with CMR imaging. *JACC Cardiovasc Imaging.* 2013;6(7):806-22.
7. Nguyen C, Fan Z, Xie Y, Dawkins J, Tseliou E, Bi X, *et al.* In vivo contrast free chronic myocardial infarction characterization using diffusion-weighted cardiovascular magnetic resonance. *J Cardiovasc Magn Reson.* 2014;16:68.
8. Wu MT, Tseng WY, Su MY, Liu CP, Chiou KR, Wedeen VJ, *et al.* Diffusion tensor magnetic resonance imaging mapping the fiber architecture remodeling in human myocardium after infarction: correlation with viability and wall motion. *Circulation.* 2006;114(10):1036-45.
9. Potet J, Rahmouni A, Mayer J, Vignaud A, Lim P, Luciani A, *et al.* Detection of myocardial edema with low-b-value diffusion-weighted echo-planar imaging sequence in patients with acute myocarditis. *Radiology.* 2013;269(2):362-9.
10. Stankovic Z, Allen BD, Garcia J, Jarvis KB, Markl M. 4D flow imaging with MRI. *Cardiovasc Diagn Ther.* 2014;4(2):173-92.
11. Holloway CJ, Suttie J, Dass S, Neubauer S. Clinical cardiac magnetic resonance spectroscopy. *Prog Cardiovasc Dis.* 2011;54(3):320-7.
12. Arnold JR, Karamitsos TD, Bhamra-Ariza P, Francis JM, Searle N, Robson MD, *et al.* Myocardial oxygenation in coronary artery disease: insights from blood oxygen level-dependent magnetic resonance imaging at 3 tesla. *J Am Coll Cardiol.* 2012;59(22):1954-64.
13. Wech T, Pickl W, Tran-Gia J, Ritter C, Beer M, Hahn D, *et al.* Whole-heart cine MRI in a single breath-hold--a compressed sensing accelerated 3D acquisition technique for assessment of cardiac function. *Rofo.* 2014;186(1):37-41.

REFERENCES

14. Herzog B, Greenwood J, Plein S. European Society of Cardiology. Cardiovascular Magnetic Resonance Pocket Guide. 2013. Available at http://www.escardio.org/static_file/Escardio/Subspecialty/EACVI/CMR-guide-2013.pdf.
15. Myerson SG, Francis J, Neubauer S. Cardiovascular Magnetic Resonance (Oxford Specialist Handbooks in Cardiology). 2013. London: Oxford University Press.
16. Society for Cardiovascular Magnetic Resonance. Available at http://www.scmr.org/.
17. 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.
18. Kramer CM, Barkhausen J, Flamm SD, Kim RJ, Nagel E. Standardized cardiovascular magnetic resonance (CMR) protocols 2013 update. *J Cardiovasc Magn Reson.* 2013;15:91.
19. Akinpelu D. Pharmacologic stress testing. Medscape. Available at http://emedicine.medscape.com/article/1827166 -overview (Updated Feb 2015).
20. Hundley WG, Bluemke D, Bogaert JG, Friedrich MG, Higgins CB, Lawson MA, *et al.* Society for Cardiovascular Magnetic Resonance guidelines for reporting cardiovascular magnetic resonance examinations. *J Cardiovasc Magn Reson.* 2009;11:5.
21. Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, *et al.* Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation.* 2002;105(4):539-42.
22. Society for Cardiovascular Magnetic Resonance (SCMR) Clinical Practice Committee. Guidelines for credentialing in cardiovascular magnetic resonance (CMR). *J Cardiovasc Magn Reson.* 2000;2(3):233-4.
23. Pohost GM, Kim RJ, Kramer CM, Manning WJ. Task Force 12: training in advanced cardiovascular imaging (cardiovascular magnetic resonance [CMR]) endorsed by the Society for Cardiovascular Magnetic Resonance. *J Am Coll Cardiol.* 2008;51(3):404-8.

REFERENCES

24. Budoff MJ, Cohen MC, Garcia MJ, Hodgson JM, Hundley WG, Lima JA, *et al.* ACCF/AHA clinical competence statement on cardiac imaging with computed tomography and magnetic resonance: a report of the American College of Cardiology Foundation/American Heart Association/American College of Physicians Task Force on Clinical Competence and Training. *J Am Coll Cardiol.* 2005;46(2):383-402.

25. Kramer CM, Budoff MJ, Fayad ZA, Ferrari VA, Goldman C, Lesser JR, *et al.* ACCF/AHA 2007 clinical competence statement on vascular imaging with computed tomography and magnetic resonance. A report of the American College of Cardiology Foundation/American Heart Association/American College of Physicians Task Force on Clinical Competence and Training. *J Am Coll Cardiol.* 2007;50(11):1097-114.

26. Sim K, Abdul Aziz Y, Chin S, Chong F, Choo G, Chew D, *et al.* The Malaysian consensus statement on utilisation of cardiac CT. *Biomed Imaging Interv J.* 2008;4(4):e41.

27. Ibrahim T, Bulow HP, Hackl T, Hornke M, Nekolla SG, Breuer M, *et al.* Diagnostic value of contrast-enhanced magnetic resonance imaging and single-photon emission computed tomography for detection of myocardial necrosis early after acute myocardial infarction. *J Am Coll Cardiol.* 2007;49(2):208-16.

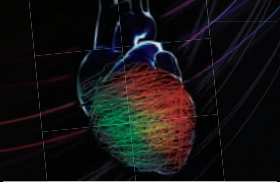
28. Kim HW, Farzaneh-Far A, Kim RJ. Cardiovascular magnetic resonance in patients with myocardial infarction: current and emerging applications. *J Am Coll Cardiol.* 2009;55(1):1-16.

29. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined--a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *J Am Coll Cardiol.* 2000;36(3):959-69.

30. Wright J, Adriaenssens T, Dymarkowski S, Desmet W, Bogaert J. Quantification of myocardial area at risk with T2-weighted CMR: comparison with contrast-enhanced CMR and coronary angiography. *JACC Cardiovasc Imaging.* 2009;2(7):825-31.

31. Lockie T, Nagel E, Redwood S, Plein S. Use of cardiovascular magnetic resonance imaging in acute coronary syndromes. *Circulation.* 2009;119(12):1671-81.

32. Gerber BL, Rousseau MF, Ahn SA, le Polain de Waroux JB, Pouleur AC, Phlips T, *et al.* Prognostic value of myocardial viability by delayed-enhanced magnetic resonance in patients with coronary artery disease and low ejection fraction: impact of revascularization therapy. *J Am Coll Cardiol.* 2012;59(9):825-35.



33. Wu KC. CMR of microvascular obstruction and hemorrhage in myocardial infarction. *J Cardiovasc Magn Reson.* 2012;14: 68.

34. Goyal P, Weinsaft JW. Cardiovascular magnetic resonance imaging for assessment of cardiac thrombus. *Methodist Debakey Cardiovasc J.* 2013;9(3):132-6.

35. Bellenger NG, Grothues F, Smith GC, Pennell DJ. Quantification of right and left ventricular function by cardiovascular magnetic resonance. *Herz.* 2000;25(4):392-9.

36. Pennell DJ, Sechtem UP, Higgins CB, Manning WJ, Pohost GM, Rademakers FE, *et al.* Clinical indications for cardiovascular magnetic resonance (CMR): Consensus Panel report. *Eur Heart J.* 2004;25(21):1940-65.

37. Maceira AM, Prasad SK, Khan M, Pennell DJ. Reference right ventricular systolic and diastolic function normalized to age, gender and body surface area from steady-state free precession cardiovascular magnetic resonance. *Eur Heart J.* 2006;27(23):2879-88.

38. Grothues F, Moon JC, Bellenger NG, Smith GS, Klein HU, Pennell DJ. Interstudy reproducibility of right ventricular volumes, function, and mass with cardiovascular magnetic resonance. *Am Heart J.* 2004;147(2):218-23.

39. Bellenger NG, Burgess MI, Ray SG, Lahiri A, Coats AJ, Cleland JG, *et al.* Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography and cardiovascular magnetic resonance; are they interchangeable? *Eur Heart J.* 2000;21(16):1387-96.

40. Di Carli MF, Maddahi J, Rokhsar S, Schelbert HR, Bianco-Batlles D, Brunken RC, *et al.* Long-term survival of patients with coronary artery disease and left ventricular dysfunction: implications for the role of myocardial viability assessment in management decisions. *J Thorac Cardiovasc Surg.* 1998;116(6):997-1004.

41. Baer FM, Theissen P, Schneider CA, Voth E, Sechtem U, Schicha H, *et al.* Dobutamine magnetic resonance imaging predicts contractile recovery of chronically dysfunctional myocardium after successful revascularization. *J Am Coll Cardiol.* 1998;31(5):1040-8.

42. 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 T1-201 tomography and dobutamine stress echocardiography. *J Am Coll Cardiol.* 2000;35(5):1152-61.

43. Beek AM, van Rossum AC. Use of cardiovascular magnetic resonance imaging in the assessment of left ventricular function, scar and viability in patients with ischaemic cardiomyopathy and chronic myocardial infarction. *Heart.* 2010;96(18):1494-501.

44. Kim RJ, Shah DJ. Fundamental concepts in myocardial viability assessment revisited: when knowing how much is "alive" is not enough. *Heart.* 2004;90(2):137-40.

45. 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.

46. Schvartzman PR, Srichai MB, Grimm RA, Obuchowski NA, Hammer DF, McCarthy PM, *et al.* Nonstress delayed-enhancement magnetic resonance imaging of the myocardium predicts improvement of function after revascularization for chronic ischemic heart disease with left ventricular dysfunction. *Am Heart J.* 2003;146(3):535-41.

47. Kirschbaum SW, Rossi A, van Domburg RT, Gruszczynska K, Krestin GP, Serruys PW, *et al.* Contractile reserve in segments with nontransmural infarction in chronic dysfunctional myocardium using low-dose dobutamine CMR. *JACC Cardiovasc Imaging.* 2010;3(6):614-22.

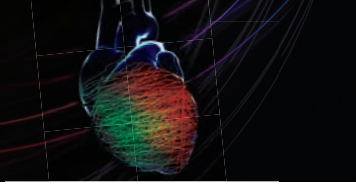
48. Kuhl HP, van der Weerdt A, Beek A, Visser F, Hanrath P, van Rossum A. Relation of end-diastolic wall thickness and the residual rim of viable myocardium by magnetic resonance imaging to myocardial viability assessed by fluorine-18 deoxyglucose positron emission tomography. *Am J Cardiol.* 2006;97(4):452-7.

49. Kwon DH, Halley CM, Carrigan TP, Zysek V, Popovic ZB, Setser R, *et al.* Extent of left ventricular scar predicts outcomes in ischemic cardiomyopathy patients with significantly reduced systolic function: a delayed hyperenhancement cardiac magnetic resonance study. *JACC Cardiovasc Imaging.* 2009;2(1):34-44.

50. Cleland JG, Daubert JC, Erdmann E, Freemantle N, Gras D, Kappenberger L, *et al.* The effect of cardiac resynchronization on morbidity and mortality in heart failure. *N Engl J Med.* 2005;352(15):1539-49.

51. Leclercq C, Kass DA. Retiming the failing heart: principles and current clinical status of cardiac resynchronization. *J Am Coll Cardiol.* 2002;39(2):194-201.

52. Chung ES, Leon AR, Tavazzi L, Sun JP, Nihoyannopoulos P, Merlino J, *et al.* Results of the Predictors of Response to CRT (PROSPECT) trial. *Circulation.* 2008;117(20):2608-16.



53. Bilchick KC, Dimaano V, Wu KC, Helm RH, Weiss RG, Lima JA, *et al.* Cardiac magnetic resonance assessment of dyssynchrony and myocardial scar predicts function class improvement following cardiac resynchronization therapy. *JACC Cardiovasc Imaging.* 2008;1(5):561-8.

54. Ypenburg C, Roes SD, Bleeker GB, Kaandorp TA, de Roos A, Schalij MJ, *et al.* Effect of total scar burden on contrast-enhanced magnetic resonance imaging on response to cardiac resynchronization therapy. *Am J Cardiol.* 2007;99(5):657-60.

55. Chalil S, Foley PW, Muyhaldeen SA, Patel KC, Yousef ZR, Smith RE, *et al.* Late gadolinium enhancement-cardiovascular magnetic resonance as a predictor of response to cardiac resynchronization therapy in patients with ischaemic cardiomyopathy. *Europace.* 2007;9(11):1031-7.

56. Lipke C, Katoh M, Franke A, Krombach G, Buecker A, Kuhl HP. The value of non-contrast harmonic transthoracic echocardiography for the detection of left ventricular thrombi in patients with cardiomyopathy: comparison with contrast-enhanced magnetic resonance imaging. *Int J Cardiovasc Imaging.* 2007;23(4):479-87.

57. Srichai MB, Junor C, Rodriguez LL, Stillman AE, Grimm RA, Lieber ML, *et al.* Clinical, imaging, and pathological characteristics of left ventricular thrombus: a comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathological validation. *Am Heart J.* 2006;152(1):75-84.

58. Wahl A, Paetsch I, Gollesch A, Roethemeyer S, Foell D, Gebker R, *et al.* Safety and feasibility of high-dose dobutamine-atropine stress cardiovascular magnetic resonance for diagnosis of myocardial ischaemia: experience in 1000 consecutive cases. *Eur Heart J.* 2004;25(14):1230-6.

59. Charoenpanichkit C, Hundley WG. The 20 year evolution of dobutamine stress cardiovascular magnetic resonance. *J Cardiovasc Magn Reson.* 2010;12:59.

60. Paetsch I, Jahnke C, Ferrari VA, Rademakers FE, Pellikka PA, Hundley WG, *et al.* Determination of interobserver variability for identifying inducible left ventricular wall motion abnormalities during dobutamine stress magnetic resonance imaging. *Eur Heart J.* 2006;27(12):1459-64.

61. Rerkpattanapipat P, Morgan TM, Neagle CM, Link KM, Hamilton CA, Hundley WG. Assessment of preoperative cardiac risk with magnetic resonance imaging. *Am J Cardiol.* 2002;90(4):416-9.

REFERENCES

62. Pereira E, Bettencourt N, Ferreira N, Schuster A, Chiribiri A, Primo J, *et al.* Incremental value of adenosine stress cardiac magnetic resonance in coronary artery disease detection. *Int J Cardiol.* 2013;168(4):4160-7.

63. Schwitter J, Wacker CM, Wilke N, Al-Saadi N, Sauer E, Huettle K, *et al.* MR-IMPACT II: Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary artery disease Trial: perfusion-cardiac magnetic resonance vs. single-photon emission computed tomography for the detection of coronary artery disease: a comparative multicentre, multivendor trial. *Eur Heart J.* 2013;34(10):775-81.

64. Doesch C, Seeger A, Doering J, Herdeg C, Burgstahler C, Claussen CD, *et al.* Risk stratification by adenosine stress cardiac magnetic resonance in patients with coronary artery stenoses of intermediate angiographic severity. *JACC Cardiovasc Imaging.* 2009;2(4):424-33.

65. Li M, Zhou T, Yang LF, Peng ZH, Ding J, Sun G. Diagnostic accuracy of myocardial magnetic resonance perfusion to diagnose ischemic stenosis with fractional flow reserve as reference: systematic review and meta-analysis. *JACC Cardiovasc Imaging.* 2014;7(11):1098-105.

66. Assomull RG, Lyne JC, Keenan N, Gulati A, Bunce NH, Davies SW, *et al.* The role of cardiovascular magnetic resonance in patients presenting with chest pain, raised troponin, and unobstructed coronary arteries. *Eur Heart J.* 2007;28(10):1242-9.

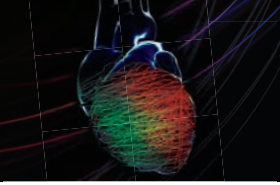
67. Kindermann I, Barth C, Mahfoud F, Ukena C, Lenski M, Yilmaz A, *et al.* Update on myocarditis. *J Am Coll Cardiol.* 2012;59(9):779-92.

68. Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, *et al.* Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol.* 2009;53(17):1475-87.

69. Friedrich MG, Marcotte F. Cardiac magnetic resonance assessment of myocarditis. *Circ Cardiovasc Imaging.* 2013;6 (5):833-9.

70. Maron BJ, Maron MS. Hypertrophic cardiomyopathy. *Lancet.* 2013;381(9862):242-55.

71. Maron BJ, McKenna WJ, Danielson GK, Kappenberger LJ, Kuhn HJ, Seidman CE, *et al.* American College of Cardiology/European Society of Cardiology clinical expert consensus document on hypertrophic cardiomyopathy. A report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *J Am Coll Cardiol.* 2003;42(9):1687-713.



72. Rubinshtein R, Glockner JF, Ommen SR, Araoz PA, Ackerman MJ, Sorajja P, *et al.* Characteristics and clinical significance of late gadolinium enhancement by contrast-enhanced magnetic resonance imaging in patients with hypertrophic cardiomyopathy. *Circ Heart Fail.* 2010;3(1):51-8.

73. Hoey ET, Elassaly M, Ganeshan A, Watkin RW, Simpson H. The role of magnetic resonance imaging in hypertrophic cardiomyopathy. *Quant Imaging Med Surg.* 2014;4(5):397-406.

74. Maron MS, Maron BJ, Harrigan C, Buros J, Gibson CM, Olivotto I, *et al.* Hypertrophic cardiomyopathy phenotype revisited after 50 years with cardiovascular magnetic resonance. *J Am Coll Cardiol.* 2009;54(3):220-8.

75. Kremastinos DT, Farmakis D. Iron overload cardiomyopathy in clinical practice. *Circulation.* 2011;124(20):2253-63.

76. Pennell DJ, Udelson JE, Arai AE, Bozkurt B, Cohen AR, Galanello R, *et al.* Cardiovascular function and treatment in beta-thalassemia major: a consensus statement from the American Heart Association. *Circulation.* 2013;128(3):281-308.

77. Tanner MA, Galanello R, Dessi C, Smith GC, Westwood MA, Agus A, *et al.* A randomized, placebo-controlled, double-blind trial of the effect of combined therapy with deferoxamine and deferiprone on myocardial iron in thalassemia major using cardiovascular magnetic resonance. *Circulation.* 2007;115(14):1876-84.

78. Carpenter JP, He T, Kirk P, Roughton M, Anderson LJ, de Noronha SV, *et al.* On T2* magnetic resonance and cardiac iron. *Circulation.* 2011;123(14):1519-28.

79. Patton N, Brown G, Leung M, Bavishi K, Taylor J, Lloyd J, *et al.* Observational study of iron overload as assessed by magnetic resonance imaging in an adult population of transfusion-dependent patients with beta thalassaemia: significant association between low cardiac T2* < 10 ms and cardiac events. *Intern Med J.* 2010;40(6):419-26.

80. Kwong RY, Falk RH. Cardiovascular magnetic resonance in cardiac amyloidosis. *Circulation.* 2005;111(2):122-4.

81. O'Donnell DH, Abbara S, Chaithiraphan V, Yared K, Killeen RP, Martos R, *et al.* Cardiac MR imaging of nonischemic cardiomyopathies: imaging protocols and spectra of appearances. *Radiology.* 2012;262(2):403-22.

82. vanden Driesen RI, Slaughter RE, Strugnell WE. MR findings in cardiac amyloidosis. *AJR Am J Roentgenol.* 2006;186(6):1682-5.

REFERENCES

83. Krombach GA, Hahn C, Tomars M, Buecker A, Grawe A, Gunther RW, *et al.* Cardiac amyloidosis: MR imaging findings and T1 quantification, comparison with control subjects. *J Magn Reson Imaging.* 2007;25(6):1283-7.

84. Rathi VK, Doyle M, Yamrozik J, Williams RB, Caruppannan K, Truman C, *et al.* Routine evaluation of left ventricular diastolic function by cardiovascular magnetic resonance: a practical approach. *J Cardiovasc Magn Reson.* 2008;10:36.

85. Yeon SB, Reichek N, Tallant BA, Lima JA, Calhoun LP, Clark NR, *et al.* Validation of in vivo myocardial strain measurement by magnetic resonance tagging with sonomicrometry. *J Am Coll Cardiol.* 2001;38(2):555-61.

86. Maceira AM, Joshi J, Prasad SK, Moon JC, Perugini E, Harding I, *et al.* Cardiovascular magnetic resonance in cardiac amyloidosis. *Circulation.* 2005;111(2):186-93.

87. Perugini E, Rapezzi C, Piva T, Leone O, Bacchi-Reggiani L, Riva L, *et al.* Non-invasive evaluation of the myocardial substrate of cardiac amyloidosis by gadolinium cardiac magnetic resonance. *Heart.* 2006;92(3):343-9.

88. Vogelsberg H, Mahrholdt H, Deluigi CC, Yilmaz A, Kispert EM, Greulich S, *et al.* Cardiovascular magnetic resonance in clinically suspected cardiac amyloidosis: noninvasive imaging compared to endomyocardial biopsy. *J Am Coll Cardiol.* 2008;51(10):1022-30.

89. Maceira AM, Prasad SK, Hawkins PN, Roughton M, Pennell DJ. Cardiovascular magnetic resonance and prognosis in cardiac amyloidosis. *J Cardiovasc Magn Reson.* 2008;10:54.

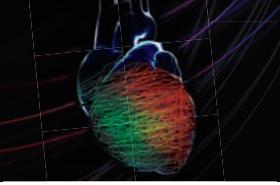
90. Perugini E, Guidalotti PL, Salvi F, Cooke RM, Pettinato C, Riva L, *et al.* Noninvasive etiologic diagnosis of cardiac amyloidosis using 99mTc-3,3-diphosphono-1,2-propanodicarboxylic acid scintigraphy. *J Am Coll Cardiol.* 2005;46(6):1076-84.

91. Srinivasan G, Joseph M, Selvanayagam JB. Recent advances in the imaging assessment of infiltrative cardiomyopathies. *Heart.* 2013;99(3):204-13.

92. Syed IS, Glockner JF, Feng D, Araoz PA, Martinez MW, Edwards WD, *et al.* Role of cardiac magnetic resonance imaging in the detection of cardiac amyloidosis. *JACC Cardiovasc Imaging.* 2010;3(2):155-64.

93. Bargout R, Kelly RF. Sarcoid heart disease: clinical course and treatment. *Int J Cardiol.* 2004;97(2):173-82.

94. Smedema JP, Snoep G, van Kroonenburgh MP, van Geuns RJ, Cheriex EC, Gorgels AP, *et al.* The additional value of gadolinium-enhanced MRI to standard assessment for cardiac involvement in patients with pulmonary sarcoidosis. *Chest.* 2005;128(3):1629-37.



95. Soejima K, Yada H. The work-up and management of patients with apparent or subclinical cardiac sarcoidosis: with emphasis on the associated heart rhythm abnormalities. *J Cardiovasc Electrophysiol.* 2009;20(5):578-83.

96. Mehta D, Lubitz SA, Frankel Z, Wisnivesky JP, Einstein AJ, Goldman M, *et al.* Cardiac involvement in patients with sarcoidosis: diagnostic and prognostic value of outpatient testing. *Chest.* 2008;133(6):1426-35.

97. 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.

98. Uemura A, Morimoto S, Hiramitsu S, Kato Y, Ito T, Hishida H. Histologic diagnostic rate of cardiac sarcoidosis: evaluation of endomyocardial biopsies. *Am Heart J.* 1999;138(2 Pt 1):299-302.

99. Smedema JP, Snoep G, van Kroonenburgh MP, van Geuns RJ, Dassen WR, Gorgels AP, *et al.* Evaluation of the accuracy of gadolinium-enhanced cardiovascular magnetic resonance in the diagnosis of cardiac sarcoidosis. *J Am Coll Cardiol.* 2005;45(10):1683-90.

100. 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.

101. Vignaux O, Dhote R, Duboc D, Blanche P, Dusser D, Weber S, *et al.* Clinical significance of myocardial magnetic resonance abnormalities in patients with sarcoidosis: a 1-year follow-up study. *Chest.* 2002;122(6):1895-901.

102. Ichinose A, Otani H, Oikawa M, Takase K, Saito H, Shimokawa H, *et al.* MRI of cardiac sarcoidosis: basal and subepicardial localization of myocardial lesions and their effect on left ventricular function. *AJR Am J Roentgenol.* 2008;191(3):862-9.

103. Basso C, Corrado D, Marcus FI, Nava A, Thiene G. Arrhythmogenic right ventricular cardiomyopathy. *Lancet.* 2009;373(9671):1289-300.

104. Rampazzo A, Nava A, Malacrida S, Beffagna G, Bauce B, Rossi V, *et al.* Mutation in human desmoplakin domain binding to plakoglobin causes a dominant form of arrhythmogenic right ventricular cardiomyopathy. *Am J Hum Genet.* 2002;71(5):1200-6.

105. McKoy G, Protonotarios N, Crosby A, Tsatsopoulou A, Anastasakis A, Coonar A, *et al.* Identification of a deletion in plakoglobin in arrhythmogenic right ventricular cardiomyopathy with palmoplantar keratoderma and woolly hair (Naxos disease). *Lancet.* 2000;355(9221):2119-24.

106. Corrado D, Thiene G. Arrhythmogenic right ventricular cardiomyopathy/dysplasia: clinical impact of molecular genetic studies. *Circulation.* 2006;113(13):1634-7.

107. Matsumori A, Furukawa Y, Hasegawa K, Sato Y, Nakagawa H, Morikawa Y, *et al.* Epidemiologic and clinical characteristics of cardiomyopathies in Japan: results from nationwide surveys. *Circ J.* 2002;66(4):323-36.

108. Marcus FI, McKenna WJ, Sherrill D, Basso C, Bauce B, Bluemke DA, *et al.* Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the Task Force Criteria. *Eur Heart J.* 2010;31(7):806-14.

109. 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.

110. Grothoff M, Pachowsky M, Hoffmann J, Posch M, Klaassen S, Lehmkuhl L, *et al.* Value of cardiovascular MR in diagnosing left ventricular non-compaction cardiomyopathy and in discriminating between other cardiomyopathies. *Eur Radiol.* 2012;22(12):2699-709.

111. Choudhary P, Hsu CJ, Grieve S, Smillie C, Singarayar S, Semsarian C, *et al.* Improving the diagnosis of LV non-compaction with cardiac magnetic resonance imaging. *Int J Cardiol.* 2015;181:430-6.

112. Bertaso AG, Richardson JD, Wong DT, Azarisman S, Teo KS, Worthley SG. An unusual cause of cardiac failure. *J Cardiovasc Med (Hagerstown).* 2012;13(3):205-6.

113. Sharkey SW, Lesser JR, Maron BJ. Cardiology Patient Page. Takotsubo (stress) cardiomyopathy. *Circulation.* 2011;124(18):e460-2.

114. Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P, Carbone I, Muellerleile K, Aldrovandi A, *et al.* Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. *JAMA.* 2011;306(3):277-86.

115. Naruse Y, Sato A, Kasahara K, Makino K, Sano M, Takeuchi Y, *et al.* The clinical impact of late gadolinium enhancement in Takotsubo cardiomyopathy: serial analysis of cardiovascular magnetic resonance images. *J Cardiovasc Magn Reson.* 2011;13:67.

116. Imbriaco M, Messalli G, Avitabile G, Cuocolo A, Maurea S, Soscia F, *et al.* Cardiac magnetic resonance imaging illustrating Anderson-Fabry disease progression. *Br J Radiol.* 2010;83(996):e249-51.

117. Desnick RJ, Brady R, Barranger J, Collins AJ, Germain DP, Goldman M, *et al.* Fabry disease, an under-recognized multisystemic disorder: expert recommendations for diagnosis, management, and enzyme replacement therapy. *Ann Intern Med.* 2003;138(4):338-46.

118. Hoey ET, Neil-Gallagher E. Utility of gadolinium enhanced cardiovascular MRI to differentiate Fabry's disease from other causes of hypertrophic cardiomyopathy. *Postgrad Med J.* 2012;88(1046):731-2.
119. Chee KH, Azman W. Prevalence and outcome of peripartum cardiomyopathy in Malaysia. *Int J Clin Pract.* 2009;63(5):722-5.
120. Givertz MM. Cardiology patient page: peripartum cardiomyopathy. *Circulation.* 2013;127(20):e622-6.
121. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, *et al.* Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. *JAMA*. 2000;283(9):1183-8.
122. Haghikia A, Röntgen P, Vogel-Claussen J, Hilfiker-Kleiner D, Bauersachs J. Characterization of peripartum cardiomyopathy by cardiovascular magnetic resonance imaging. *J Cardiovasc Magn Reson.* 2015;17(Suppl 1):Q46.
123. Marmursztejn J, Vignaux O, Goffinet F, Cabanes L, Duboc D. Delayed-enhanced cardiac magnetic resonance imaging features in peripartum cardiomyopathy. *Int J Cardiol.* 2009;137(3):e63-4.
124. Kawano H, Tsuneto A, Koide Y, Tasaki H, Sueyoshi E, Sakamoto I, *et al.* Magnetic resonance imaging in a patient with peripartum cardiomyopathy. *Intern Med.* 2008;47(2):97-102.
125. Mouquet F, Lions C, de Groote P, Bouabdallaoui N, Willoteaux S, Dagorn J, *et al.* Characterisation of peripartum cardiomyopathy by cardiac magnetic resonance imaging. *Eur Radiol.* 2008;18(12):2765-9.
126. Elliott P. Diagnosis and management of dilated cardiomyopathy. *Heart* 2000;84:106–112.
127. Awtry EH, Philippides GJ. Alcoholic and cocaine-associated cardiomyopathies. *Prog Cardiovasc Dis.* 2010;52(4):289-99.
128. Piano MR, Phillips SA. Alcoholic cardiomyopathy: pathophysiologic insights. *Cardiovasc Toxicol.* 2014;14(4): 291-308.
129. McCrohon JA, Moon JC, Prasad SK, McKenna WJ, Lorenz CH, Coats AJ, *et al.* Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. *Circulation.* 2003;108(1):54-9.
130. Kim DH, Choi SI, Chang HJ, Choi DJ, Lim C, Park JH. Delayed hyperenhancement by contrast-enhanced magnetic resonance imaging: Clinical application for various cardiac diseases. *J Comput Assist Tomogr.* 2006;30(2):226-32.
131. Moller S, Henriksen JH. Cardiovascular complications of cirrhosis. *Gut.* 2008;57(2):268-78.

132. Powell AJ, Tsai-Goodman B, Prakash A, Greil GF, Geva T. Comparison between phase-velocity cine magnetic resonance imaging and invasive oximetry for quantification of atrial shunts. *Am J Cardiol.* 2003;91(12):1523-5.

133. Thomson LE, Crowley AL, Heitner JF, Cawley PJ, Weinsaft JW, Kim HW, *et al.* Direct en face imaging of secundum atrial septal defects by velocity-encoded cardiovascular magnetic resonance in patients evaluated for possible transcatheter closure. *Circ Cardiovasc Imaging.* 2008;1(1):31-40.

134. Steffens JC, Bourne MW, Sakuma H, O'Sullivan M, Higgins CB. Quantification of collateral blood flow in coarctation of the aorta by velocity encoded cine magnetic resonance imaging. *Circulation.* 1994;90(2):937-43.

135. Grosse-Wortmann L, Al-Otay A, Yoo SJ. Aortopulmonary collaterals after bidirectional cavopulmonary connection or Fontan completion: quantification with MRI. *Circ Cardiovasc Imaging.* 2009;2(3):219-25.

136. Rathod RH, Prakash A, Kim YY, Germanakis IE, Powell AJ, Gauvreau K, *et al.* Cardiac magnetic resonance parameters predict transplantation-free survival in patients with fontan circulation. *Circ Cardiovasc Imaging.* 2014;7(3):502-9.

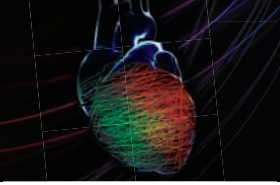
137. Attenhofer Jost CH, Edmister WD, Julsrud PR, Dearani JA, Savas Tepe M, Warnes CA, *et al.* Prospective comparison of echocardiography versus cardiac magnetic resonance imaging in patients with Ebstein's anomaly. *Int J Cardiovasc Imaging.* 2012;28(5):1147-59.

138. Yalonetsky S, Tobler D, Greutmann M, Crean AM, Wintersperger BJ, Nguyen ET, *et al.* Cardiac magnetic resonance imaging and the assessment of ebstein anomaly in adults. *Am J Cardiol.* 2011;107(5):767-73.

139. Mostbeck GH, Caputo GR, Higgins CB. MR measurement of blood flow in the cardiovascular system. *AJR Am J Roentgenol.* 1992;159(3):453-61.

140. Lin SJ, Brown PA, Watkins MP, Williams TA, Lehr KA, Liu W, *et al.* Quantification of stenotic mitral valve area with magnetic resonance imaging and comparison with Doppler ultrasound. *J Am Coll Cardiol.* 2004;44(1):133-7.

141. Kupfahl C, Honold M, Meinhardt G, Vogelsberg H, Wagner A, Mahrholdt H, *et al.* Evaluation of aortic stenosis by cardiovascular magnetic resonance imaging: comparison with established routine clinical techniques. *Heart.* 2004;90(8):893-901.



142. Reant P, Lederlin M, Lafitte S, Serri K, Montaudon M, Corneloup O, *et al.* Absolute assessment of aortic valve stenosis by planimetry using cardiovascular magnetic resonance imaging: comparison with transesophageal echocardiography, transthoracic echocardiography, and cardiac catheterisation. *Eur J Radiol.* 2006;59(2):276-83.

143. Kon MW, Myerson SG, Moat NE, Pennell DJ. Quantification of regurgitant fraction in mitral regurgitation by cardiovascular magnetic resonance: comparison of techniques. *J Heart Valve Dis.* 2004;13(4):600-7.

144. Dulce MC, Mostbeck GH, O'Sullivan M, Cheitlin M, Caputo GR, Higgins CB. Severity of aortic regurgitation: interstudy reproducibility of measurements with velocity-encoded cine MR imaging. *Radiology.* 1992;185(1):235-40.

145. Cawley PJ, Maki JH, Otto CM. Cardiovascular magnetic resonance imaging for valvular heart disease: technique and validation. *Circulation.* 2009;119(3):468-78.

146. Aurigemma G, Reichek N, Schiebler M, Axel L. Evaluation of aortic regurgitation by cardiac cine magnetic resonance imaging: planar analysis and comparison to Doppler echocardiography. *Cardiology.* 1991;78(4):340-7.

147. Nishimura T, Yamada N, Itoh A, Miyatake K. Cine MR imaging in mitral regurgitation: comparison with color Doppler flow imaging. *AJR Am J Roentgenol.* 1989;153(4):721-4.

148. Kilner PJ, Manzara CC, Mohiaddin RH, Pennell DJ, Sutton MG, Firmin DN, *et al.* Magnetic resonance jet velocity mapping in mitral and aortic valve stenosis. *Circulation.* 1993;87(4):1239-48.

149. Caruthers SD, Lin SJ, Brown P, Watkins MP, Williams TA, Lehr KA, *et al.* Practical value of cardiac magnetic resonance imaging for clinical quantification of aortic valve stenosis: comparison with echocardiography. *Circulation.* 2003;108(18):2236-43.

150. John AS, Dill T, Brandt RR, Rau M, Ricken W, Bachmann G, *et al.* Magnetic resonance to assess the aortic valve area in aortic stenosis: how does it compare to current diagnostic standards? *J Am Coll Cardiol.* 2003;42(3):519-26.

151. Schlosser T, Malyar N, Jochims M, Breuckmann F, Hunold P, Bruder O, *et al.* Quantification of aortic valve stenosis in MRI-comparison of steady-state free precession and fast low-angle shot sequences. *Eur Radiol.* 2007;17(5):1284-90.

152. Heidenreich PA, Steffens J, Fujita N, O'Sullivan M, Caputo GR, Foster E, *et al.* Evaluation of mitral stenosis with velocity-encoded cine-magnetic resonance imaging. *Am J Cardiol.* 1995;75(5):365-9.

153. Djavidani B, Debl K, Lenhart M, Seitz J, Paetzel C, Schmid FX, *et al.* Planimetry of mitral valve stenosis by magnetic resonance imaging. *J Am Coll Cardiol.* 2005;45(12):2048-53.

154. Djavidani B, Debl K, Buchner S, Lipke C, Nitz W, Feuerbach S, *et al.* MRI planimetry for diagnosis and follow-up of valve area in mitral stenosis treated with valvuloplasty. *Rofo.* 2006;178(8):781-6.

155. Wintersperger BJ, Becker CR, Gulbins H, Knez A, Bruening R, Heuck A, *et al.* Tumors of the cardiac valves: imaging findings in magnetic resonance imaging, electron beam computed tomography, and echocardiography. *Eur Radiol.* 2000;10(3):443-9.

156. Sievers B, Brandts B, Franken U, Trappe HJ. Cardiovascular magnetic resonance imaging demonstrates mitral valve endocarditis. *Am J Med.* 2003;115(8):681-2.

157. Leiner T, Kessels AG, Nelemans PJ, Vasbinder GB, de Haan MW, Kitslaar PE, *et al.* Peripheral arterial disease: comparison of color duplex US and contrast-enhanced MR angiography for diagnosis. *Radiology.* 2005;235(2):699-708.

158. Ouwendijk R, de Vries M, Pattynama PM, van Sambeek MR, de Haan MW, Stijnen T, *et al.* Imaging peripheral arterial disease: a randomized controlled trial comparing contrast-enhanced MR angiography and multi-detector row CT angiography. *Radiology.* 2005;236(3):1094-103.

159. Kramer U, Nael K, Laub G, Nyborg GK, Fenchel M, Miller S, *et al.* High-resolution magnetic resonance angiography of the renal arteries using parallel imaging acquisition techniques at 3.0 T: initial experience. *Invest Radiol.* 2006;41(2):125-32.

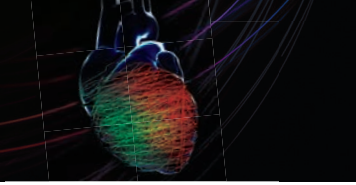
160. Nael K, Saleh R, Lee M, McNamara T, Godinez SR, Laub G, *et al.* High-spatial-resolution contrast-enhanced MR angiography of abdominal arteries with parallel acquisition at 3.0 T: initial experience in 32 patients. *AJR Am J Roentgenol.* 2006;187(1):W77-85.

161. Fussen S, De Boeck BW, Zellweger MJ, Bremerich J, Goetschalckx K, Zuber M, *et al.* Cardiovascular magnetic resonance imaging for diagnosis and clinical management of suspected cardiac masses and tumours. *Eur Heart J.* 2011;32(12):1551-60.

162. Kaminaga T, Takeshita T, Kimura I. Role of magnetic resonance imaging for evaluation of tumors in the cardiac region. *Eur Radiol.* 2003;13(Suppl 6):L1-10.

163. Varghese A, Pennell DJ. Cardiovascular Magnetic Resonance Made Easy. Churchill Livingston. 2008.

164. Bogaert J, Francone M. Cardiovascular magnetic resonance in pericardial diseases. *J Cardiovasc Magn Reson.* 2009;11:14.



REFERENCES

165. Staab W, Schwarz A, Menke J, Spiro JE, Zwaka PA, Lotz J, *et al.* Prevalence and clinical relevance of extra-cardiac findings at cardiac magnetic resonance imaging. *J Cardiovasc Magn Reson.* 2013;15(Suppl 1):P267.

166. Irwin RB, Newton T, Peebles C, Borg A, Clark D, Miller C, *et al.* Incidental extra-cardiac findings on clinical CMR. *Eur Heart J Cardiovasc Imaging.* 2013;14(2):158-66.

ACKNOWLEDGEMENTS

The Writing Committee for the Consensus Statement on the Utilisation of Cardiac Magnetic Resonance 2015 would like to acknowledge the Ministry of Health Malaysia, the National Heart Association of Malaysia and the College of Radiology, Academy of Medicine of Malaysia for their contribution in the preparation of this document.

Our sincerest appreciation to Dr. Noor Hisham Abdullah, Director General of Health, Malaysia; Dr. Rosli Mohd Ali, President of the National Heart Association of Malaysia; and, Dr. Abdul Rahman Mohamad, President of the College of Radiology, Academy of Medicine of Malaysia in taking the time to pen down the opening messages for this inaugural Consensus Statement.

We would also like to thank the President and the staff of Heart House, National Heart Association of Malaysia for access to the meeting rooms, use of the facilities and refreshments during the committee meetings.

National Heart Association of Malaysia

Level 1, Heart House
Medical Academies of Malaysia
210, Jalan Tun Razak
50400 Kuala Lumpur
Malaysia