

**Series Editors**

**Bernhard A. Herzog**

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EUROPEAN ASSOCIATION  
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IMAGING

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**EUROPEAN  
SOCIETY OF  
CARDIOLOGY®**

# **Cardiovascular Magnetic Resonance Pocket Guide**

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**Second Edition May 2017**



# Foreword

**Cardiovascular Magnetic Resonance (CMR) is now an established diagnostic procedure with a wide range of clinical indications. This pocket guide aims to provide a brief overview of the use of CMR in routine clinical practice. It provides a summary of the most commonly used CMR methods, clinical applications and normal ranges for CMR measurements. The pocket guide is intended for day-to-day use on the scanner or reporting station and provides typical scan protocols, tips and tricks and guidance for reporting for the most common clinical applications of CMR. This second edition of the pocket guide has been enriched with many more case examples and a new section on the differential diagnoses of common CMR findings.**

**Bernhard Herzog**

**John Greenwood**

**Sven Plein**

The Cardiovascular Magnetic Resonance Pocket Guide represents the views of the authors and the European Association of Cardiovascular Imaging. It was arrived at after careful consideration of the available evidence at the time it was written. Health professionals are encouraged to take it fully into account when exercising their clinical judgment. This pocket guide does not, however, override the individual responsibility of health professionals to make appropriate decisions in the circumstances of the individual patients, in consultation with that patient and, where appropriate and necessary, the patient's guardian or carer. It is also the health professional's responsibility to verify the applicable rules and regulations applicable to drugs and devices at the time of prescription.

We acknowledge the support and advice we have received from Regina Herzog, Gavin Bainbridge, Ananth Kidambi, Manish Motwani, Akhlaque Uddin, Filip Zemrak, Juliano de Lara Fernandes, Maciej Garbowksi and Amna Abdel-Gadir.

# Table of Contents

<b>1</b>	<b>Standard views</b>	<b>5</b>	<ul style="list-style-type: none"><li>• Stress perfusion imaging</li></ul>	<b>28</b>
<b>2</b>	<b>Image quality</b>	<b>12</b>	<ul style="list-style-type: none"><li>• Dobutamine stress imaging</li></ul>	<b>31</b>
	<ul style="list-style-type: none"><li>• Difficult Imaging</li></ul>	<b>12</b>	<ul style="list-style-type: none"><li>• Transmurality of myocardial infarction</li></ul>	<b>33</b>
	<ul style="list-style-type: none"><li>• Artefacts</li></ul>	<b>13</b>	<ul style="list-style-type: none"><li>• Fatty replacement in myocardial infarction</li></ul>	<b>34</b>
<b>3</b>	<b>Components of CMR protocols</b>	<b>15</b>	<b>5</b> <b>Acute myocardial infarction</b>	<b>35</b>
	<ul style="list-style-type: none"><li>• Anatomy, LV and RV function</li></ul>	<b>15</b>	<b>6</b> <b>Anomalous coronary arteries</b>	<b>37</b>
	<ul style="list-style-type: none"><li>• Oedema</li></ul>	<b>16</b>	<b>7</b> <b>Cardiomyopathies</b>	<b>39</b>
	<ul style="list-style-type: none"><li>• T2★ Mapping</li></ul>	<b>16</b>	Dilated cardiomyopathy	<b>39</b>
	<ul style="list-style-type: none"><li>• T1 Mapping</li></ul>	<b>17</b>	Hypertrophic cardiomyopathy	<b>41</b>
	<ul style="list-style-type: none"><li>• Tagging</li></ul>	<b>20</b>	Infiltrative cardiomyopathies	<b>43</b>
	<ul style="list-style-type: none"><li>• Angiography</li></ul>	<b>20</b>	<ul style="list-style-type: none"><li>• Amyloidosis</li></ul>	<b>43</b>
	<ul style="list-style-type: none"><li>• Coronary Artery Imaging</li></ul>	<b>21</b>	<ul style="list-style-type: none"><li>• Anderson Fabry disease</li></ul>	<b>47</b>
	<ul style="list-style-type: none"><li>• Phase Velocity Encoded Flow</li></ul>	<b>22</b>	<ul style="list-style-type: none"><li>• Iron overload cardiomyopathy</li></ul>	<b>49</b>
	<ul style="list-style-type: none"><li>• Perfusion</li></ul>	<b>23</b>	Sarcoidosis	<b>51</b>
	<ul style="list-style-type: none"><li>• Gadolinium enhancement</li></ul>	<b>24</b>	Tako-Tsubo Cardiomyopathy	<b>53</b>
<b>4</b>	<b>Chronic coronary heart disease</b>	<b>25</b>	Non Compaction Cardiomyopathy	<b>55</b>
	<ul style="list-style-type: none"><li>• Coronary supply</li></ul>	<b>25</b>	Arrhythmogenic Right Ventricular Cardiomyopathy	<b>58</b>
	<ul style="list-style-type: none"><li>• The 17 segment model</li></ul>	<b>27</b>	Becker and Duchenne Muscular Dystrophy	<b>62</b>

# Table of Contents

Endomyocardial fibrosis	64	LGE patterns	93
<b>8 Myocarditis</b>	<b>65</b>	• LV recesses and outpouchings	<b>98</b>
<b>9 Pericardial disease</b>	<b>67</b>	• LV hypertrophy	<b>100</b>
• Pericarditis	67	• RV dilatation	<b>102</b>
• Constrictive pericarditis	68	<b>14 Normal values</b>	<b>106</b>
• Pericardial effusion	70	• Male LV reference values	<b>106</b>
<b>10 Valvular heart disease</b>	<b>72</b>	• Female LV reference values	<b>107</b>
• Mitral valve imaging	73	• Male RV reference values	<b>108</b>
• Mitral regurgitation	74	• Female RV reference values	<b>109</b>
• Mitral stenosis	76	• Atrial reference values	<b>110</b>
• Aortic valve imaging	77	• Aortic root reference values	<b>111</b>
• Aortic regurgitation	78	<b>15 Safety</b>	<b>112</b>
• Aortic stenosis	79	• Gadolinium contrast agents	<b>112</b>
• Tricuspid and pulmonary valves	80	• Common devices	<b>113</b>
<b>11 Aortic disease</b>	<b>81</b>	<b>16 Miscellaneous</b>	<b>114</b>
• Aortic diameters	82	• Common MR terminology	<b>114</b>
<b>12 Cardiac masses</b>	<b>85</b>	• Abbreviations	<b>115</b>
<b>13 Differential diagnosis</b>	<b>93</b>	• References	<b>116</b>

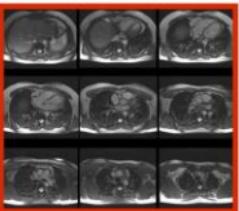
# Standard Views - Localizers



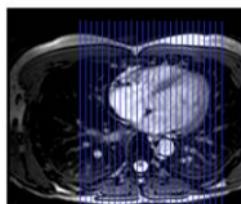
Sagittal localizer



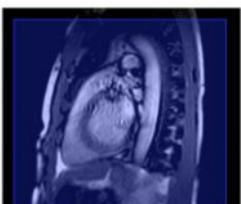
Transaxial localizer



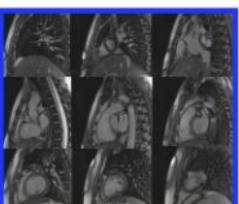
Transaxial stack



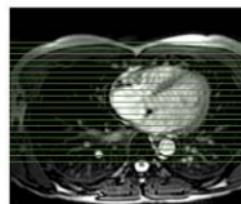
Transaxial localizer



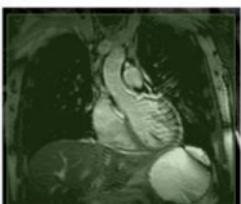
Sagittal localizer



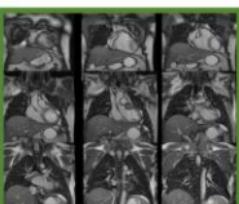
Sagittal stack



Transaxial localizer



Coronal localizer



Coronal stack

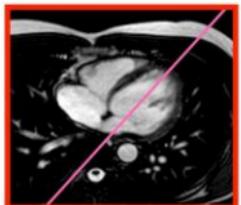
## Localizers

- A CMR study begins with acquisition of a stack of localizer scans
- Localizers are acquired in transaxial, sagittal and coronal orientations
- Localizers are used to plan additional scan planes
- They are typically acquired with low resolution and free breathing
- Localizers allow a first overview of cardiac anatomy and planning of subsequent views

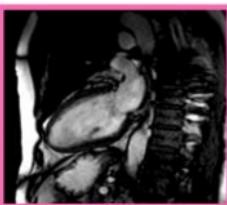
## Tips & Tricks

- **Check if patient is in the isocentre of the magnet:** ensure sufficient signal in the heart
- **Check coil element selection:** ensure signal seen from all relevant coil elements.

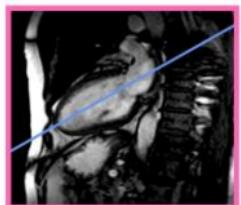
# Standard Views - Left Ventricle



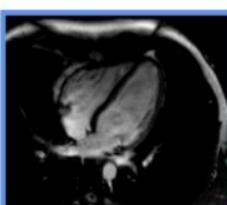
Transaxial stack



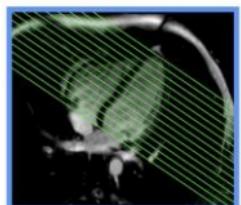
p2CH



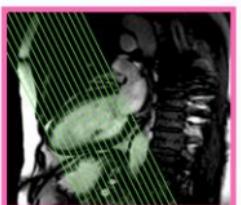
p2CH



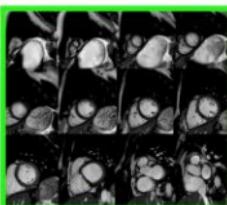
p4CH



p4CH



p2CH

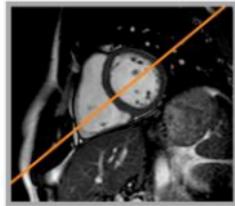


SA Stack

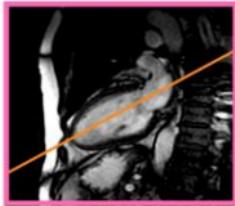
## Obtaining LV *pseudo*- views

- Planning of *pseudo*-views uses the localizer images obtained previously
- Pseudo 2 chamber (p2CH) view (also called Vertical Long Axis view):** is obtained by positioning a plane perpendicular to the transaxial plane through the LV apex and mitral valve (this is an oblique 2 chamber plane rather than a true perpendicular one)
- Pseudo 4 chamber (p4CH) view (also called Horizontal Long Axis view):** is obtained from the p2CH by cutting an orthogonal plane through the apex and MV (this is also not yet a true 4CH view)
- True short axis (SA) view:** is obtained by cutting a plane orthogonal to both the p2CH and p4CH views
- Typically a **stack of SA** cines covering the entire LV is then obtained

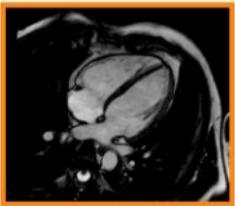
# Standard Views - Left Ventricle



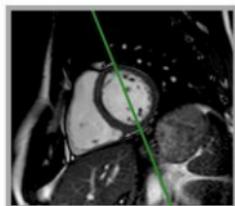
SA



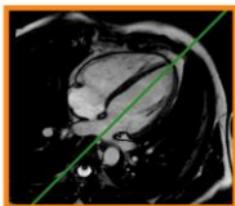
p2CH or p4CH



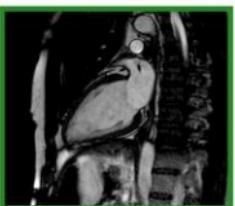
4CH\*



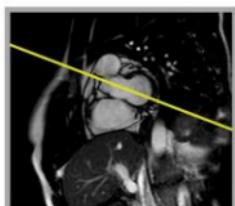
SA



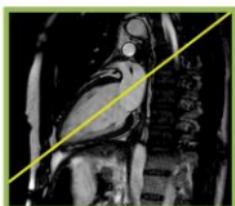
4CH



2CH



SA base



2CH or 4CH



3CH

## Obtaining LV true-views

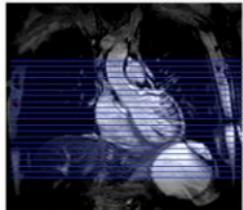
- **4CH view:** is obtained by planning a plane orthogonal to the SA view and the p2CH view, dissecting the RV at its maximal lateral dimension and the LV below the antero-lateral papillary muscle
- **2CH view:** is obtained by planning a plane orthogonal to the p4CH or 4CH view and the SA view, dissecting the inferior and anterior LV wall parallel to the septum
- **3CH view:** is obtained by planning a plane orthogonal to the 2CH or 4CH view and the basal SA view, dissecting the aortic valve and posterior wall

## Tips & Tricks

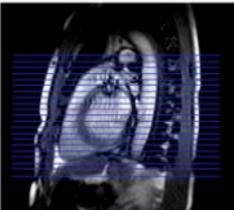
- **Simplified planning:** Use p2CH and p4CH instead of 2CH and 4CH
- **Synonyms:** 2CH=VLA; 4CH=HLA; 3CH=Sagittal LVOT

\*A dilated aortic root may remain in view even in 4CH

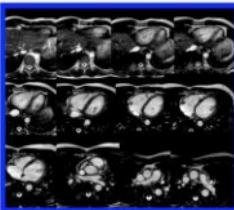
# Standard Views - Right Ventricle



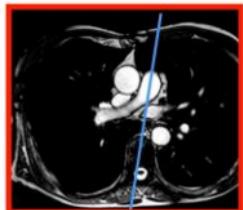
Coronal localizer



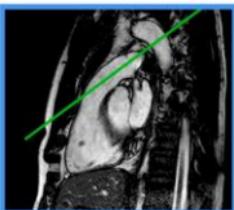
Sagittal localizer



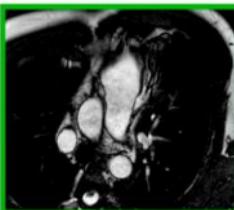
RV stack°



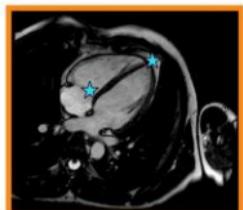
Transaxial stack



Sagittal RVOT



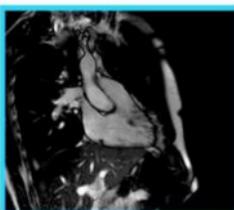
Coronal RVOT



4CH



Sagittal RVOT



RV in-/outflow

★ 3-point planning

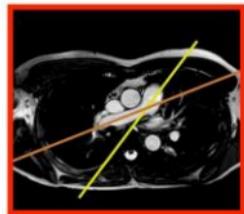
## Obtaining standard RV views

- **RV transaxial stack:** can be obtained in a transaxial plane from the level of the diaphragm to the pulmonary bifurcation or as a stack of 4CH cines
- **Sagittal RVOT:** is obtained by planning a sagittal plane directly through the main pulmonary artery, the pulmonary valve and RV cavity
- **Coronal RVOT:** is obtained by planning an orthogonal plane through the sagittal RVOT image
- **RV in-/outflow:** is best obtained by 3-point planning. The 3 points are placed on the tricuspid valve, the RV apex in 4CH view and the pulmonary valve in the RVOT view

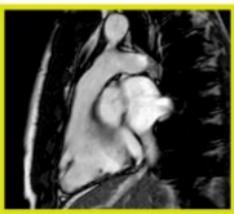
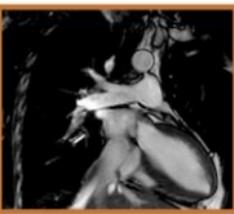
## Tips & Tricks

- For RV transaxial stack the slice positions can be copied from the transaxial localizer
- RV measurements can also be made from the SA LV stack.

# Standard Views - Pulmonary Arteries

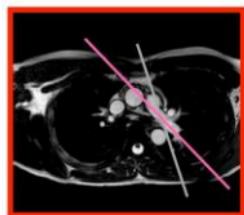


Transaxial stack

Proximal RPA<sup>+</sup>Distal RPA<sup>+</sup>

## Obtaining standard PA views

- **Left and right PA:** are obtained by planning orthogonal planes through the respective pulmonary arteries in transaxial views
- Different angulations of these planes provide **proximal vs distal** views of both pulmonary arteries



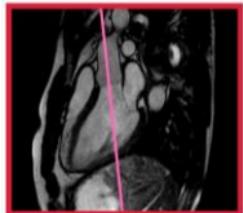
Transaxial stack

Proximal LPA<sup>+</sup>Distal LPA<sup>+</sup>

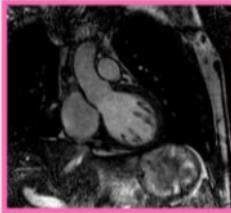
\* Full bifurcation planned from transaxial and coronal localizer

# Standard Views - Aortic Valve & Aorta

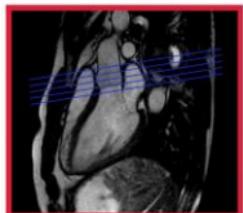
10



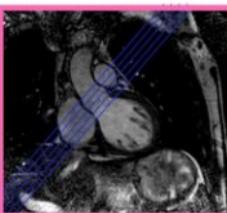
3CH view



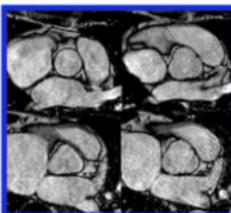
Coronal LVOT



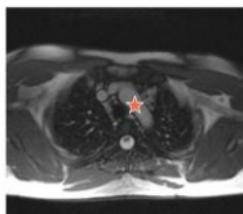
3CH view



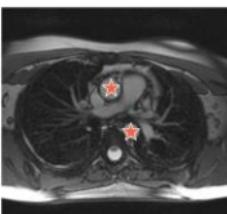
Coronal LVOT



Aortic valve /root



Transaxial



Transaxial



Aortic arch

## Obtaining standard aortic valve views

- **Coronal LVOT/AV view:** is obtained by prescribing an orthogonal plane through the aortic root in the 3CH view
- **In plane AV view:** is planned perpendicular to aortic root and parallel to aortic valve in 3CH and coronal LVOT views

## Obtaining standard aorta views

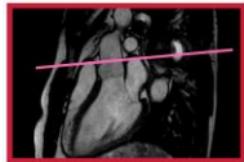
- **Aortic arch:** use the 3-point planning technique – in the transaxial stack set one point in the ascending, one point in the descending aorta and one point at the level of the aortic arch

## Tips & Tricks

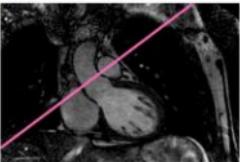
- Acquire several in plane AV slices (usually 4-5)
- Also review axial images for assessment of the aorta

★ 3-point planning

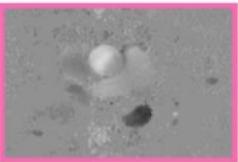
# Standard Views - Flow Aorta & Pulmonary Arteries



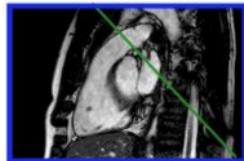
Sagittal LVOT



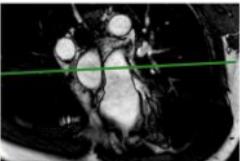
Coronal LVOT



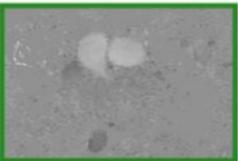
Ao flow



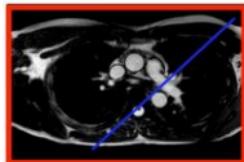
Sagittal RVOT



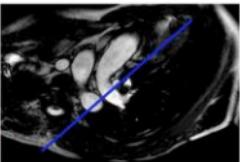
Coronal RVOT



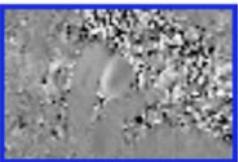
MPA flow



Transaxial stack



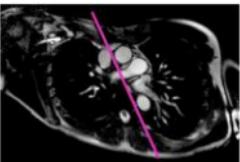
LPA cross-cut



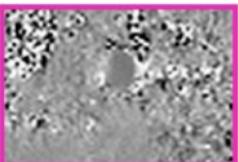
LPA flow°



Transaxial stack



RPA cross-cut



RPA flow°

## Obtaining standard flow views

- **Aortic flow view:** is obtained by prescribing a plane perpendicular to the aortic root in both sagittal and coronal LVOT views
- **Main pulmonary artery (MPA) flow view:** is obtained by prescribing a plane perpendicular to MPA in both sagittal and coronal RVOT views
- **Left PA flow view:** is obtained by prescribing a plane perpendicular to the left PA in both transaxial view and its cross-cut plane
- **Right PA flow view:** is obtained by prescribing a plane perpendicular to the right PA in both transaxial view and its cross-cut plane

## Tips & Tricks

- **Simplified planning:** directly from coronal and axial localizers
- The position of the aortic flow view relative to the AV may affect aortic regurgitation measurements

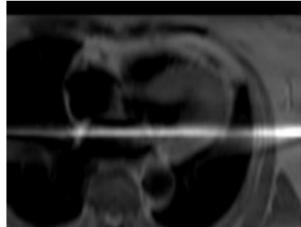
# Difficult Imaging

## Dealing with poor breath-holders

Acceleration technique	Comment
• Reduce <b>number of slices</b> acquired per breath-hold	• Increases overall scan time
• Reduce <b>number of phase encoding steps</b>	
Partial Fourier k <sub>y</sub> ( <b>Halfscan</b> )	• Reduces SNR
Increase <b>voxel size</b>	• Decreases spatial resolution
Use <b>parallel imaging</b>	• Reduces SNR, can cause artefacts
• Use <b>respiratory navigator</b>	• Increases overall scan time
• Acquire images in <b>inspiration</b>	• More variation in slice position between breath-holds
• Consider <b>general anaesthesia</b>	

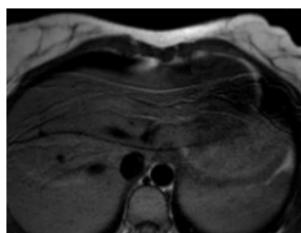
## Dealing with arrhythmias

Technique	Comment
• <b>Heart rate and/or rhythm control</b> before scanning	• Use beta-blockers or other antiarrhythmic medication
• Use <b>Arrhythmia Rejection</b>	• Increases breath-hold time
• Use <b>Prospective triggering</b>	• Decrease SNR; the end of diastole is not acquired
• Use <b>Real-time imaging</b>	• Reduces temporal and spatial resolution as well as SNR



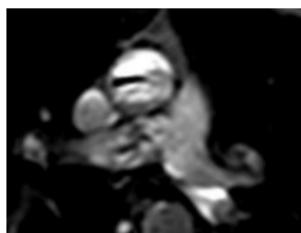
#### Wrapping artefact (fold-over, back-folding)

- Increase FOV
- Add phase encoding steps (phase-oversampling, foldover suppression, no phase wrap)
- Swap phase and frequency direction
- Use selective tissue saturation bands
- Use a surface coil
- Select only relevant coil elements



#### Ghosting artefact from motion (respiratory)

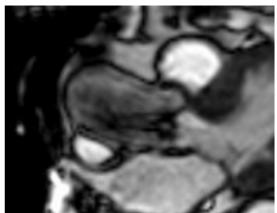
- Strict breath-holding plus acceleration techniques
- Respiratory gating or navigator echoes
- Swap phase and frequency direction
- Use selective tissue saturation bands to suppress the signal from the anterior abdominal wall



#### Ghosting artefact from motion (pulsatile flow)

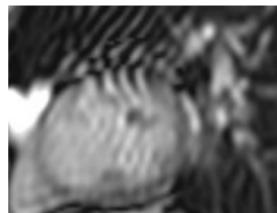
- Use ECG triggering / gating
- Use flow compensation
- Use selective tissue saturation bands to suppress the blood signal
- Swap phase and frequency direction

# Artefacts



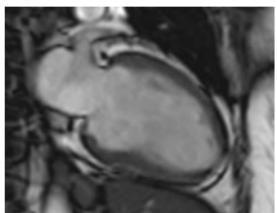
## Flow-related signal loss and flow jets

- Reduce echo time
- Use flow compensation
- Use unbalanced sequences



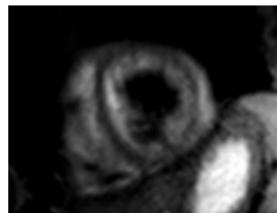
## Radiofrequency interference artefact

- Check for sources of interference and eliminate (e.g. make sure scan room door is closed)



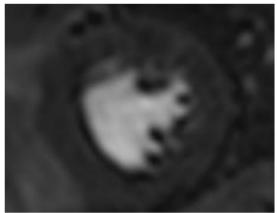
## Chemical shift artefact

- Compare with other images as they are sequence dependent



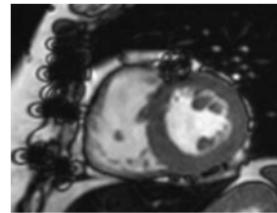
## Slow flow artefact

- Usually in T2w images
- Increase black blood pre-pulse slice thickness



## Dark rim artefact

- Often seen in perfusion imaging
- Reduce contrast dose/infusion speed
- Increase in-plane spatial resolution



## Metal artefact

- Usually less prominent on spin echo images than gradient echo images
- Avoid balanced SSFP imaging

## Anatomy stack

1. **T1w** axial black blood imaging (diaphragm to above aortic arch)
2. Free breathing or breath-hold (high resolution)
3. Slice thickness: 8-10mm (contiguous)

## LV Function

1. **Cine SSFP** pulse sequence (parallel imaging as required)
2. **2CH, 4CH, SA** and **LVOT** (2 orthogonal) cine images
3. **SA cine stack** (from mitral valve to apex)
  - Slice thickness 6-10mm
  - Inter-slice gap 0-4mm to equal 10mm
4. Temporal resolution  $\leq 45\text{ms}$

## RV Function

1. **Cine SSFP** pulse sequence (parallel imaging as required)
2. **Trans-axial cine stack** (from diaphragm to pulmonary bifurcation) or **SA cine stack** as for LV
  - Slice thickness 6-8mm
  - Inter-slice gap 0mm
3. Temporal resolution  $\leq 45\text{ms}$

## Tips & Tricks

1. Acquire the **anatomy stack** in diastole to reduce motion artefact
2. To reduce breath-hold times use **acceleration techniques**
3. **Contouring:**
  - In a healthy heart there is usually one less slice to contour in end-systole at the base of the heart (longitudinal shortening). Correlate SA to long axis view if available to identify atrio-ventricular valve plane
  - Use the movie function of the analysis software for correct alignments
  - Different methods have been proposed to deal with trabeculation and papillary muscles. Use a consistent approach and the correct normal values for the chosen method
  - **RV volumes** are more reproducible when calculated from an axial imaging plane

### Description

1. T2w images are oedema sensitive
2. Acquire **prior to contrast** administration
3. **Slice thickness:**
  - $\geq 10\text{mm}$  to ensure good SNR
  - Slice thickness of the dark blood pre-pulse should be greater than the longitudinal shortening of the LV
4. **Mid-diastolic readout**

### Tips & Tricks

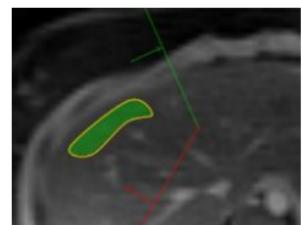
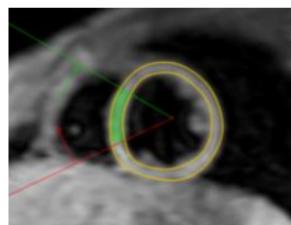
1. Use **body coil** or alternatively **functional surface coil** intensity correction algorithms to correct for coil-related signal differences
2. Slow flow artefacts may cause high signal at endocardial border
3. Consider T2 mapping for oedema detection and quantification (if available)

### Description

1. **Single breath-hold, multi-echo, T2★ sequence** (gradient echo or modified black blood sequence)
2. **Single mid-ventricular slice**
3. **Single transaxial slice of the liver**

### Tips & Tricks

1. Ensure good patient **breath-holding** by coaching as the scan duration is long
2. Make sure **the septum is of good image quality** as this is where quantification is most reproducible
3. Position the transverse liver slice correctly:
  - Avoid large hepatic vessels for correct T2\* measurement in the liver tissue



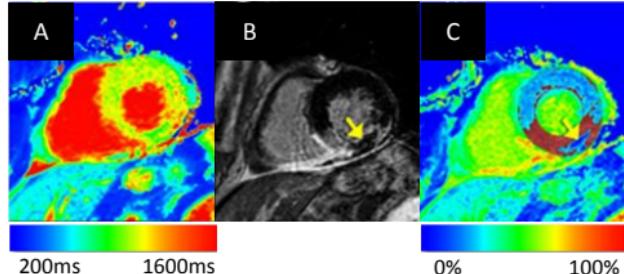
ROIs are placed in the ventricular septum (A) and the liver (B)

**Description**

1. **Scout imaging** as per LV function
2. Appropriate **vendor recommended** optimum T1-mapping sequence (e.g. MOLLI/ShMOLLI/SASHA etc.)
3. SA images - single mid-ventricular slice or 3 slices planned using “**3 of 5**” technique
4. **Gadolinium injection** (0.1 – 0.2mmol/kg)
5. Wait at least 15 min post contrast injection to acquire **post-contrast T1**
6. Take full blood count for haematocrit to calculate **extracellular volume (ECV)**

**Tips & Tricks**

1. Acquire **one slice per breath hold**
2. Native T1 values are reproducible but **they vary by magnet strength** (with 3T resulting in longer native T1 times), **vendor platforms and the mapping sequence**
3. Check scanner generated native T1 map for **breathing artefact**. Consider changing voxel size, fold-over or adding SENSE to reduce breath-hold
4. **FOV and voxel size should not be changed** between pre/post contrast T1 acquisition to allow generation of ECV maps



**Multi-parametric tissue characterisation at mid-slice in a case of acute re-perfused ST-elevation myocardial infarction affecting the inferior wall**

- **T1 mapping (A):** Native T1-values are raised in the area of risk (>1000ms) and also in the remote myocardium
- **LGE imaging (B):** inferior infarction with an area of microvascular obstruction (yellow arrow)
- **ECV mapping (C):** ECV is raised in the infarct zone but low in the MVO as this area does not take up any contrast (yellow arrow) (red areas represent ECV greater than 30%)

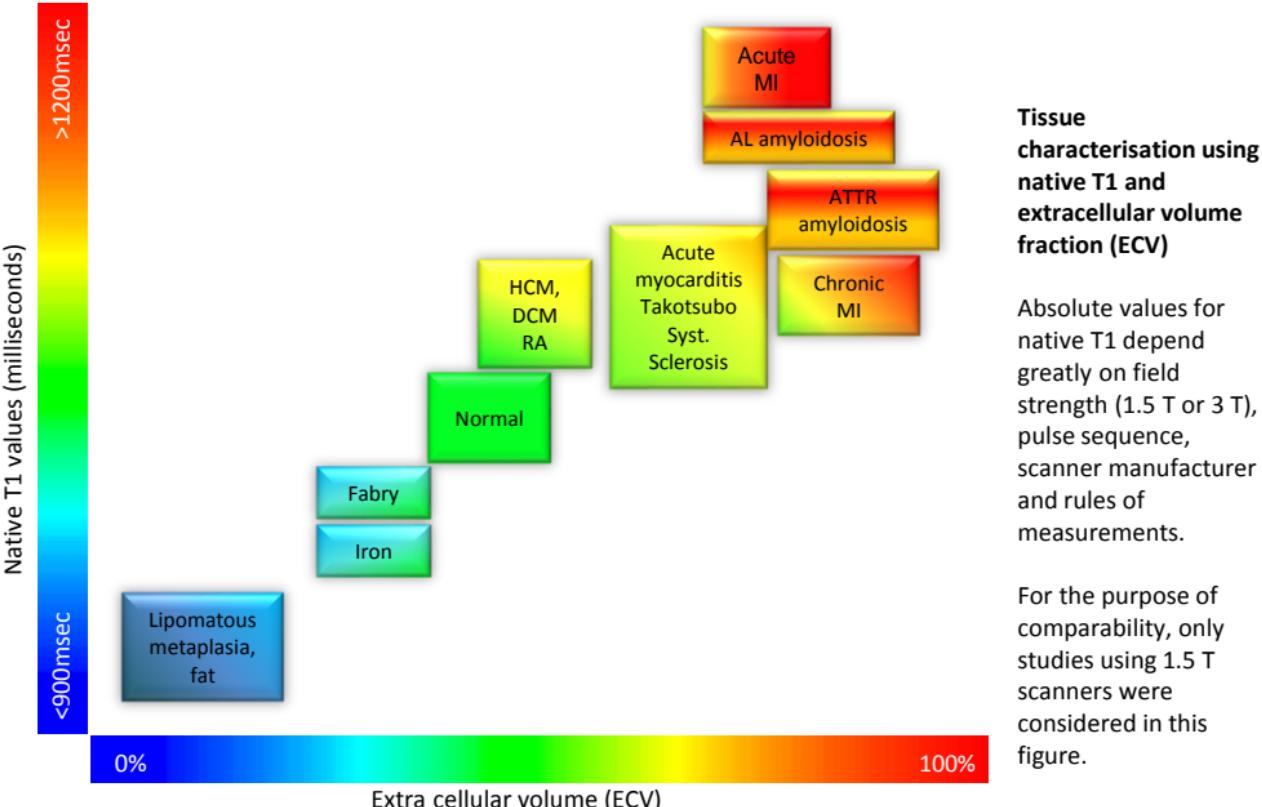
$$\text{ECV} = (1 - \text{haematocrit}) \frac{\frac{1}{\text{post contrast T1 myo}} - \frac{1}{\text{native T1 myo}}}{\frac{1}{\text{post contrast T1 blood}} - \frac{1}{\text{native T1 blood}}}$$

# T1 & T2 Mapping at a glance

18

Native T <sub>1</sub> ↑	Native T <sub>1</sub> ↓
<ul style="list-style-type: none"><li><i>Oedema</i> e.g. tissue water↑ in acute MI, inflammation, pericardial effusion</li><li><i>Increase of interstitial space</i> e.g. (replacement) fibrosis, scar, cardiomyopathy, amyloid deposition</li></ul>	<ul style="list-style-type: none"><li><i>Fat/Lipid overload</i> e.g. lipomatous metaplasia in chronic MI, Anderson-Fabry, lipoma</li><li><i>Iron overload</i></li></ul>
T <sub>2</sub> ↑	T <sub>2</sub> ↓
<ul style="list-style-type: none"><li><i>Oedema</i> e.g. tissue water↑ in acute MI, inflammation, pericardial effusion (T<sub>2</sub> more sensitive than native T<sub>1</sub> for oedema detection)</li></ul>	<ul style="list-style-type: none"><li><i>Fat/Lipid overload</i></li><li><i>Iron overload (T<sub>2</sub>*↓)</i></li></ul>

# Tissue characterisation with T1 Mapping and ECV



## Description

1. **Scout imaging** as per LV function
2. Choose **line tagging or grid tagging** pattern
3. Choose **slice orientation** from cine study
4. Acquire data in **breath-hold**

## Tips & Tricks

1. Reference modality for evaluating multidimensional strain
2. **Temporal resolution about 15-20ms**
3. **Acceleration techniques** used to shorten the breath-hold time are the same as for cine imaging
4. Use a low **flip angle** to reduce tissue saturation and prolong the tagging pattern throughout the cardiac cycle
5. Mid-myocardial circumferential strain from SA is most reproducible



Apical (A), mid-ventricular (B) and basal (C) grid-tagging

## Description

1. Prepare infusion pump with contrast agent and flush
  - Gadolinium dose: 0.1–0.2mmol/kg
2. Define **3D target region** (usually a very large volume)
3. Define required **timing of acquisition** (arterial / venous)
4. Determine best **timing parameters** for data acquisition (pre-bolus or automatic triggering)
5. Perform a **dummy** acquisition
6. Perform **acquisition** with contrast administration

## Tips & Tricks

1. Optimize timing technique:
  - Ensure that the centre of k space is acquired at the same time as the bolus of contrast arrives in the vessel of interest
2. Ensure that the FOV covers the whole area of interest including any collateral or aberrant vessels

## Description

1. Determine coronary **rest period**
  - Acquire **2CH** with **high temporal resolution** (50 phases) to determine coronary rest period
2. **Navigator-gated, free-breathing 3D pulse sequence:**
  - **Trans-axial stack**(from the proximal main pulmonary artery to the middle of the right atrium; entire cardiac coverage if desired) **or sagittal stack**
  - Slice thickness: 1-1.5 mm
  - Spatial resolution in-plane: 1.0 mm or less
  - Number of slices: typically 50 – 80
  - **Adjust trigger delay and acquisition window** according to observed coronary artery rest period
  - **Parallel acquisition** preferred
  - Navigator placed over the right hemidiaphragm
3. **Optional:**
  - Consider contrast to increase vessel conspicuity
  - T2-prepared sequence may be useful

- Breath-hold techniques if poor image quality or if navigators are unavailable or are of poor quality

## Tips & Tricks

1. **Coronary rest period:**
  - Repeat high temporal resolution 4CH scan at the correct heart rate (HR)
  - **Consider cine scan during free-breathing** if HR changes during breath-hold
  - As a compromise, scan **with longest trigger delay and a tight window** (<50 ms)
  - Coronary rest period may differ between LCA and RCA
2. **High HR ( $\geq 90$ bpm)**
  - Use shortest scan window possible to minimize blurring
  - Consider scanning at systolic rest period
3. Keep scan times to a sensible limit
5. Higher spatial resolution equals longer scan times

# Phase Velocity Encoded Flow

## Description

1. Choose the appropriate imaging plane **perpendicular to direction of flow**
2. Consider orthogonal acquisition to define peak velocity
3. Set required **direction of flow**
4. Choose **appropriate velocity encoding (VENC)**:
  - Normal aortic flow: 150cm/s
  - Normal pulmonary flow: 100cm/s
  - Adjust in pathological situations (severe valve stenosis > 400cm/s)
5. Choose adequate **spatial resolution**
  - minimum of 4-6 pixels per vessel diameter

## Tips & Tricks

1. **VENC settings:**
  - Optimal within 25% of the true peak velocity
    - Too low: flow aliasing
    - Too high: underestimating velocity
  - Correct direction of flow (Right-Left, Foot-Head)
  - Image plane distal from valve leaflet tips
  - **Flow assessment:** perpendicular to the vessel
  - **Maximal velocity assessment:** perpendicular to the jet
2. **Avoid underestimation of velocities.** Check adequate temporal resolution (phases)
  - Free-breathing acquisition: 30 phases
  - Breath-hold acquisition: 20-25 phases
3. **Rotate FOV** - orthogonal to the direction of flow
4. **Slice thickness: <7mm**

**Description**

1. **Scout imaging** as per LV function
2. **Saturation-recovery gradient echo pulse sequence**
3. **Parallel imaging** (typical two-fold acceleration)
4. **Acquisition in SA plane** (at least three slices), plus optional long axis planes
  - Slice thickness 8-10mm
  - In-plane resolution < 2.5mm
  - Ideally obtain data every heart beat
5. **Contrast (0.05 - 0.1mmol/kg, rate: 3 - 7ml/s)** followed by 30ml saline flush (3-7ml/s)
6. **Breath-hold** starts during early phases of contrast infusion **before contrast reaches the LV cavity**
7. Image for **>40 heartbeats**

**Tips & Tricks**

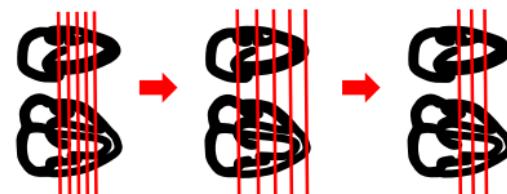
1. **Always perform a “dummy” scan** to check
  - Correct slice positioning
  - Artefacts
2. Switch to alternate heartbeat acquisition if HR is too high or reduce number of slices

**3. Field of View**

- As small as possible
- Parallel to the anterior chest wall

**4. Use “3 out of 5” technique to position slices****“3 out of 5” technique**

- Plan in end-systole in long axis views (2CH and 4CH)
- Plan 5 slices perpendicular to the longitudinal axis and adjust the slice gap so that the first slice is in the plane of the mitral valve and the last slice is at the apex.
- Remove the outer slices (1<sup>st</sup> and 5<sup>th</sup>) by reducing the number of slices from 5 to 3
- The resulted 3 slices are in accurate and reproducible positions and can be used for perfusion assessment



# Early/Late Gadolinium Enhancement

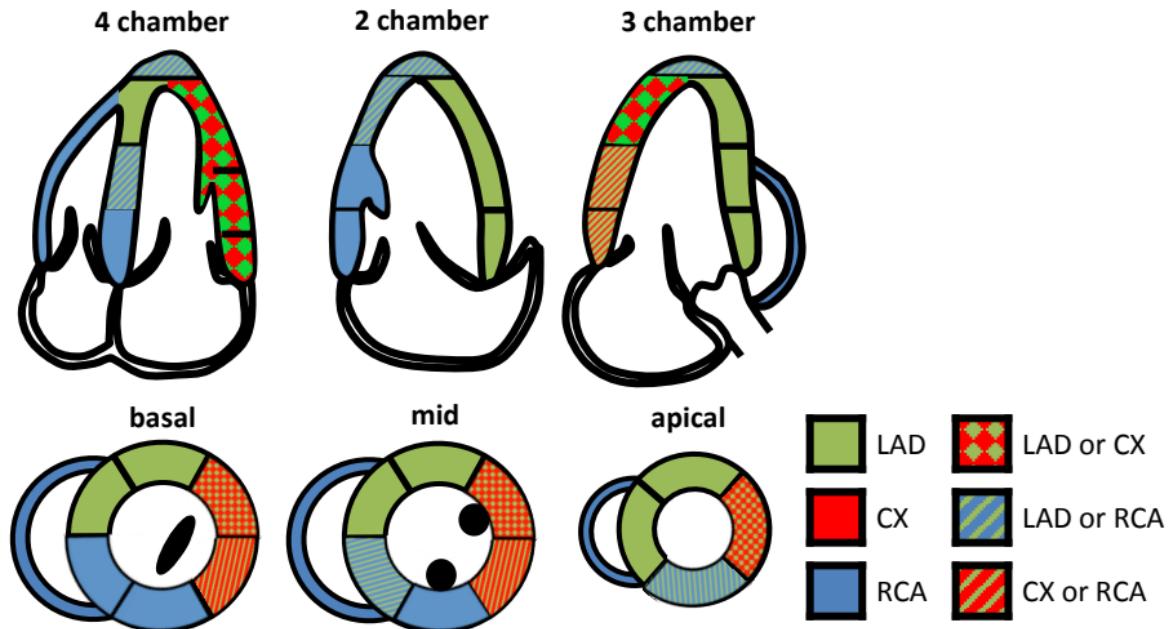
24

## Description

1. **Segmented IR GRE** (inversion recovery gradient echo) imaging during **diastolic** rest period
2. **4CH, 3CH, 2CH, SA** images
3. In-plane resolution : <2mm
4. Contrast dose 0.1 – 0.2mmol /kg
4. **Early Gadolinium Enhancement (EGE)**: acquire **1-3 min** after contrast,  $\text{TI} > 400\text{ms}$
5. **Late Gadolinium Enhancement (LGE)**: acquire  $\geq 10\text{min}$  after contrast injection
  - Consider imaging earlier if lower Gd doses are used
  - Consider imaging later in a low output state
6. **TI set to null normal myocardium:**
  - TI scout or Look Locker sequence
  - Phase-sensitive sequence with fixed TI as alternative
7. **Readout:**
  - Usually every other heartbeat
  - Every heartbeat in the setting of bradycardia
  - Every third heartbeat in the setting of tachycardia

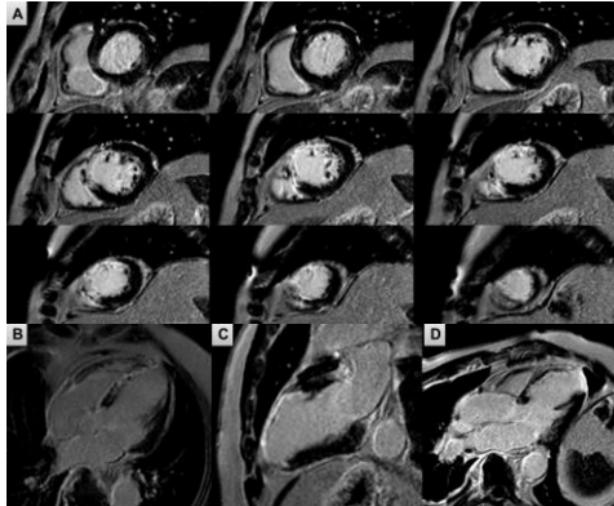
## Tips & Tricks

1. **Scan in mid- or late-diastole** to minimize motion artefacts
2. Use **saturation bands** across the spinal column and the anterior chest wall to reduce ghosting artefacts
3. **Late enhancement on images:**
  - Use “**Phase Swap**” (changing the phase encoding direction) to confirm pathology/detect artefact
  - Always consider a different plane cross-cutting through the enhanced area
4. **Increase TI times by 10 – 15ms every few minutes or repeat Look Locker scout regularly**, because the correct TI for “nulling” of normal myocardium changes over time
5. **To reduce breath-hold times use acceleration techniques**
6. Acquiring the images during every second or third heartbeat can help if there are problems with arrhythmia
7. Consider infiltrative disease (**amyloidosis**) if normal myocardium is hard to null despite correct technique

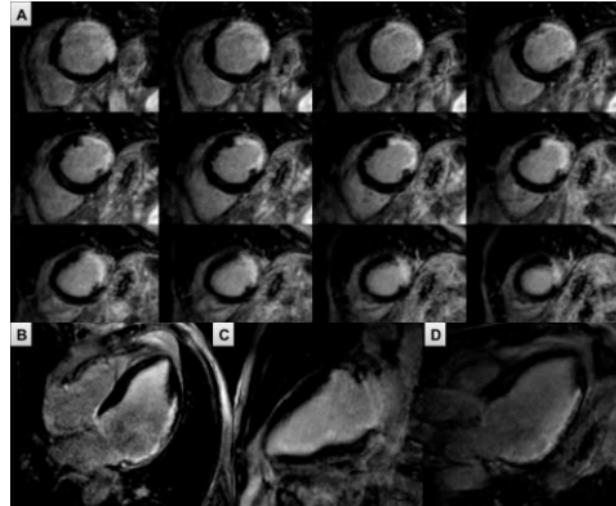


# Coronary Supply

26

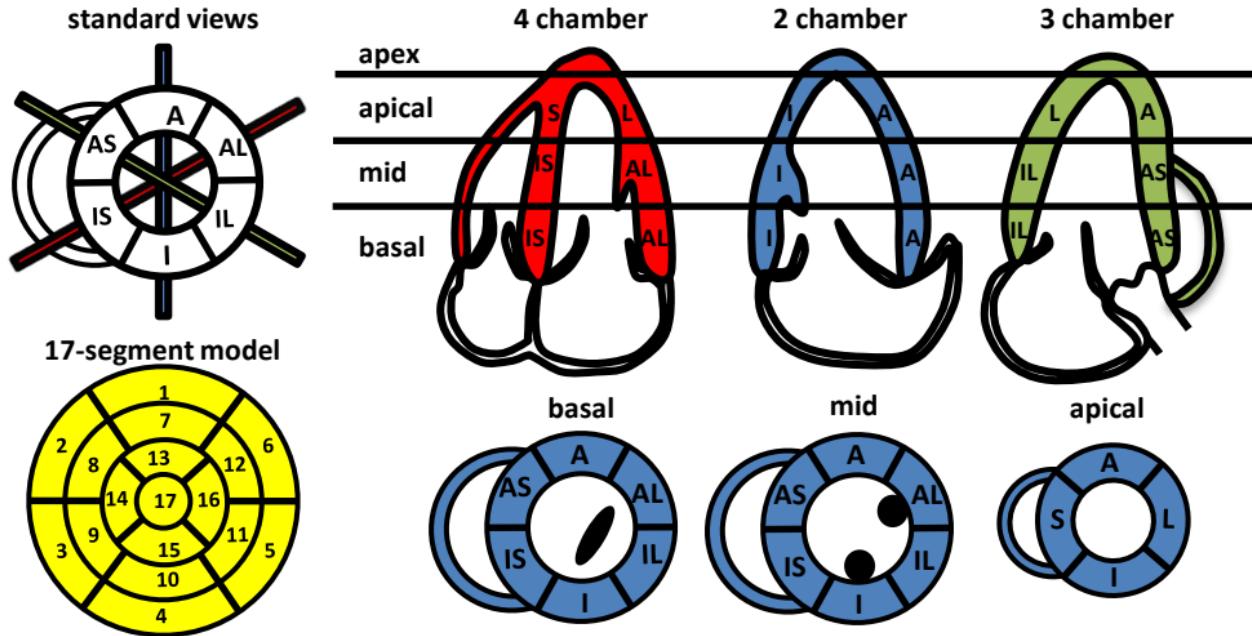


**LGE SA stack (A), 4CH (B), 2CH (C), 3CH (D):**  
Transmural myocardial infarction of the  
anteroseptal (midventricular), anterior  
(midventricular to apical) and septal (apical) wall as  
well as the apex, corresponding to the **LAD**  
**territory.**



**LGE SA stack (A), 4CH (B), 2CH (C), 3CH (D):**  
Transmural myocardial infarction of the  
anterolateral and inferolateral (basal to  
midventricular) as well as lateral (apical) wall, which  
corresponds to the **CX territory.**

# The 17 Segment AHA Model



1: basal anterior A; 2: basal anteroseptal AS; 3: basal inferoseptal IS; 4: basal inferior I; 5: basal inferolateral IL; 6: basal anterolateral AL; 7: mid-anterior A; 8: mid-antерoseptal AS; 9: mid-inferoseptal IS; 10: mid-inferior I; 11: mid-inferolateral IL; 12: mid-anterolateral AL; 13: apical anterior A; 14: apical septal S; 15: apical inferior I; 16: apical lateral L; 17: apex

# Stress Myocardial Perfusion Imaging

28

## Protocol

1. Anatomy
2. Myocardial perfusion "dummy" without contrast injection (to check position and artefact)
3. Myocardial perfusion STRESS
4. LV function
5. Myocardial perfusion REST (optional)
6. LGE

## Report

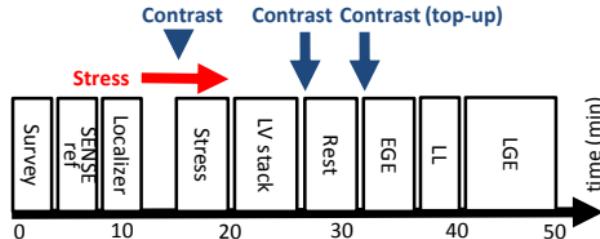
1. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF / RV: EDV, ESV, SV, EF
  - Regional wall motion abnormalities (17 segments)
2. Presence and transmural extent of scar
  - ≤25%, 26-50%, 51-75%, 76-100%
3. Presence and transmural extent of stress perfusion defect
4. Correlation between scar and perfusion defect
5. Comment on potential suitability of revascularization based on ischaemia and viable myocardium
6. (Presence and location of artefacts)

## Vasodilator Stress agents

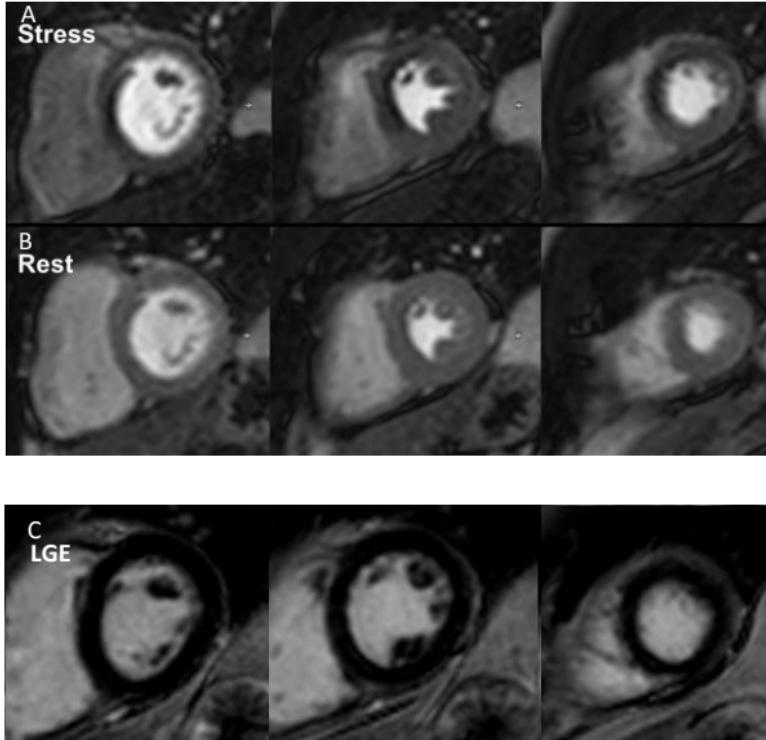
1. Adenosine:
  - Standard dose 140mcg/kg/min at least 3 min
  - Consider 170 or 210mcg/kg/min, if hemodynamic response is inadequate
2. Regadenoson 0.4 mg single injection
3. Dipyridamole 0.56 mg/kg in 4 minutes
4. Contraindications for all vasodilators: known hypersensitivity, 2nd /3rd AV nodal block, severe reversible obstructive airways disease

## Tips & Tricks

1. Low contrast uptake in spleen at first pass suggests adequate adenosine effect ('splenic switch off').
2. Regadenoson does not cause splenic switch off
3. Dobutamine may be used for stress imaging but causes high heart rate and ischaemia



# Stress Perfusion Imaging

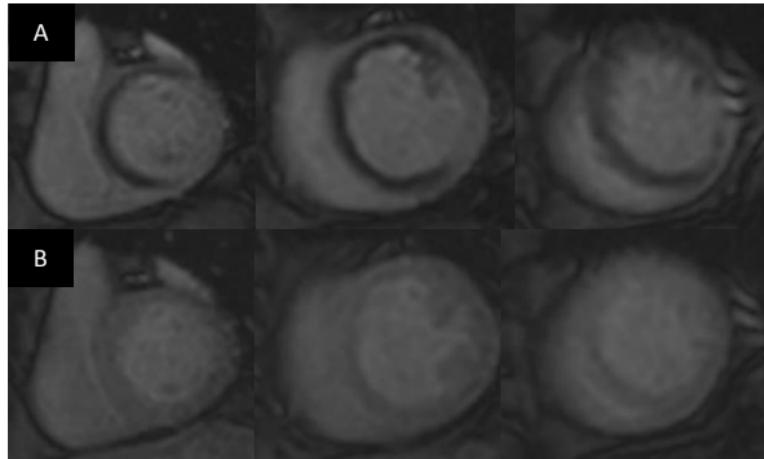


64 year old male with typical angina

- **Adenosine stress perfusion (A):** extensive perfusion defect in the anterior and anteroseptal segments
- **Rest perfusion (B):** normal
- **LGE (C):** normal signal suppression in the myocardium
- **Report:** Extensive ischaemia in the LAD territory affecting 6/17 myocardial segments (approximately 30% of myocardium)

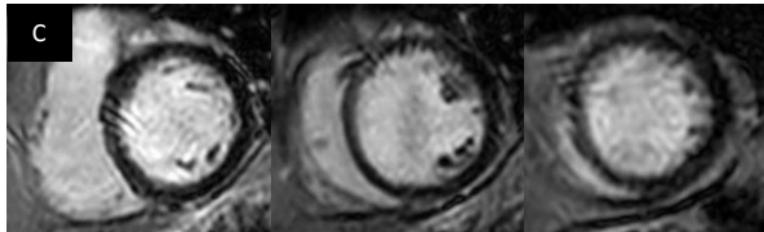
# Stress Myocardial Perfusion Imaging

30



## 72-year-old woman with exercise angina

- **Adenosine stress perfusion (A):** extensive perfusion defect in the anterior, antero-septal, infero-septal and inferior walls from base to apex (10/17 segments).
- **Rest perfusion (B):** normal
- **LGE (C):** no evidence of myocardial infarction or focal fibrosis
- **Report:** extensive and prognostically significant inducible ischaemia in the LAD and RCA territories which are entirely viable and likely to recover function following revascularisation (likely hibernation)



**Protocol**

1. Anatomy
2. LV function - 3 SA, 2-3 long axis views
3. Dobutamine Stress
  - 3min-intervals: 10 / 20 / 30 / 40 mcg/kg/min
  - HR target =  $0.85 \times (220\text{-age})$
  - Consider 0.5 mg atropine x 2 to increase HR
  - Repeat cine images at each stress level
4. LGE

**Report**

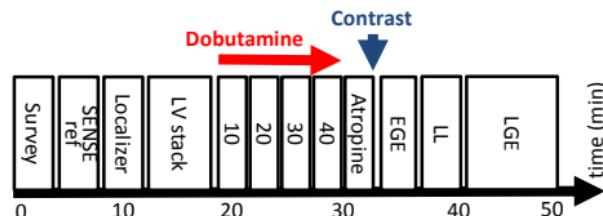
1. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF / RV: EDV, ESV, SV, EF
  - **Regional wall motion abnormalities** (17 segments)
  - **Improvement during low-dose stress** (=viability)
  - **Improvement or biphasic response during high-dose stress** (=ischaemia)
2. Presence and transmural extent of scar
3. Summarize: **resting function, contractile reserve, wall motion index, ischaemia for coronary territories**
4. Comment if any **valvular regurgitation** worsens

**Key Issues**

1. **Check BP at each stage of protocol / monitor ECG**
2. **Always view cine loops during stress online**
3. **Stop test, if any of the following occurs:**
  - New wall motion abnormalities
  - Serious side effects
  - Achievement of peak HR
4. **Contraindications for atropine:** narrow-angle glaucoma, myasthenia gravis, obstructive uropathy, obstructive gastrointestinal disorders

**Tips & Tricks**

1. Use "3 out of 5" technique to position SA slices
2. Consider acquiring perfusion images at peak stress



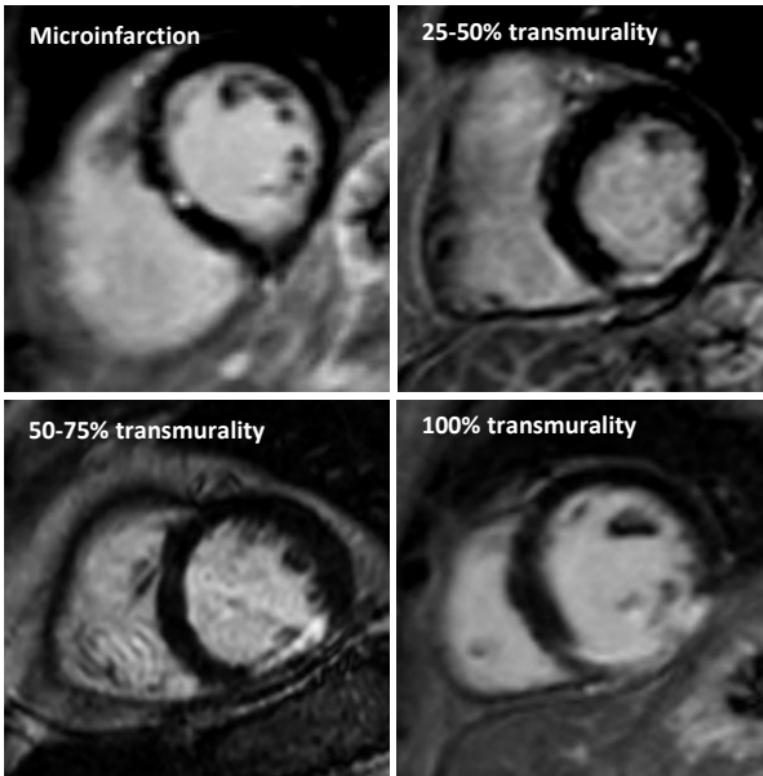
# Dobutamine Stress Imaging

32

Ischaemic Conditions	Myocardial Wall Motion			Wall Motion Score Index (WMSI) (sum of wall motion scores / number of segments)
	Rest	Low-Dose Dobutamine	High-Dose Dobutamine	
Normal				Normal 1
Ischaemia				Hypokinetic 2
Hibernation				Akinetic 3
Subendocardial scar				Dyskinetic 4
Transmural scar				Aneurysmal 5

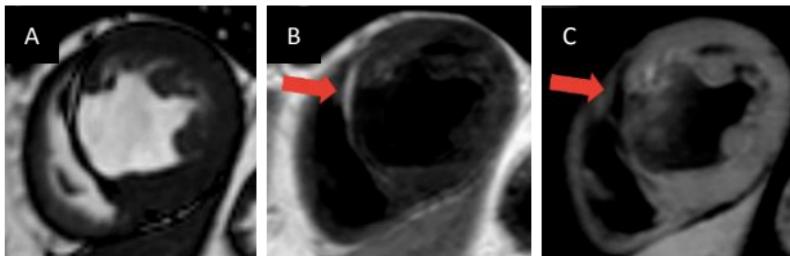
A WMSI of 1 is considered normal

# Transmurality of myocardial infarction



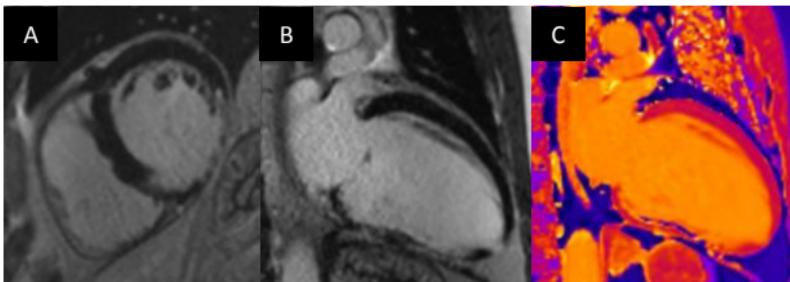
# Fatty replacement in chronic myocardial infarction

34



60-year-old man with a chronic LAD infarction

- **Cine SA (A):** thinned and akinetic anteroseptal wall
- **T1w black blood SA (B):** high signal within the thinned septum indicating fatty replacement
- **T1w black blood image with fat suppression SA (C):** signal suppression in the same area



Chronic RCA infarction in a 75-year-old woman

- **LGE SA (A) and 2CH (B):** infarction of the inferior wall
- **Native T1 mapping 2CH (C):** low native T1 values in the inferior wall suggestive of fatty replacement

**Protocol**

1. Anatomy
2. LV function
3. Oedema
4. EGE – LGE
5. T2 mapping (if available) for detection of oedema

**Report**

1. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF / RV: EDV, ESV, SV, EF
  - Regional wall motion abnormalities
2. Presence of oedema (=area at risk)
3. Presence and transmural extent of scar
4. Presence and extent of microvascular obstruction

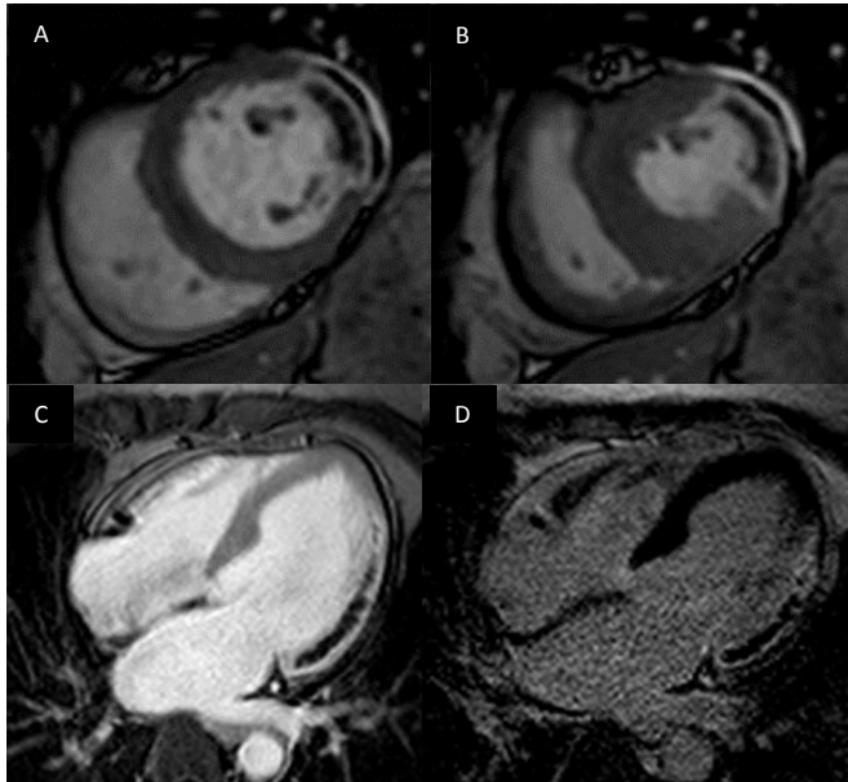
**Key Points**

1. T2w imaging may differentiate acute from chronic myocardial infarction
2. MVO:
  - Equates to angiographic “no reflow” appearance
  - High risk feature
3. Risk assessment:
  - Infarction size
  - LV / RV function
  - MVO
4. Assessment of LV thrombus on EGE images

**Tips & Tricks**

1. MVO best seen on EGE images at TI > 400ms
2. T2w images must be acquired before contrast administration
3. Compare LGE images with cine images if unsure about differentiation between blood pool and endocardial late enhancement

# Acute myocardial infarction



**45-year-old man with lateral STEMI due to circumflex artery occlusion**

- Diastolic (**A**) and systolic (**B**) phases of a **cine SA** dataset showing akinesia of the lateral wall
- **EGE 4CH (C)** showing microvascular obstruction in the basal to mid lateral wall
- **LGE SA (D)** demonstrating microvascular obstruction of the lateral wall

## Protocol

### 1. Coronary artery imaging

- 3D MRA (free breathing navigator gated)
- Targeted 2D MRA

## Report

### 1. Origin

- High / low / commissural
- From opposite coronary sinus
- Outside coronary sinuses
- Separate ostium for LAD and CX

### 2. Anomalous course

- Inter-arterial, retro-aortic, etc

### 3. Anomalies of intrinsic coronary arterial anatomy

- Ectasia, aneurysm, hypoplasia
- Intramural coronary artery (muscular bridge)

### 4. Anomalies of coronary termination

### 5. Anomalous collateral vessels

## Key Points

1. Spatial resolution can be less than that required to assess coronary lumen
2. **Malignant course:**
  - Inter-arterial course between aorta and RVOT, particular left coronary artery from right sinus
3. **Possible causes of ischaemia:**
  - Inter-arterial dynamic compression
  - Slit-like origin
  - Myocardial bridging

## Tips & Tricks

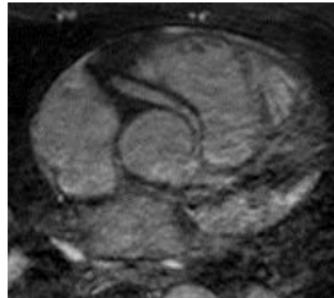
1. Optimize image quality:
  - Use isotropic voxel sizes
  - Short acquisition window (< 150ms)
2. Consider dobutamine stress (rather than adenosine) to demonstrate a regional wall motion abnormality (if inter-arterial course)

# Anomalous Coronary Arteries

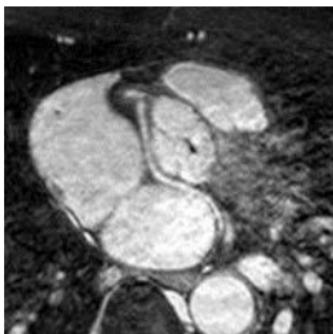
38



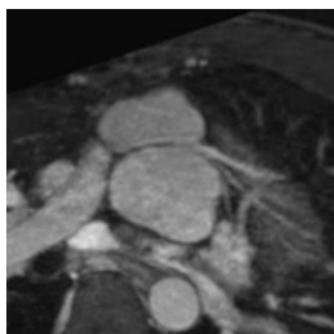
LCA from right sinus with  
inter-arterial course



RCA from left sinus with  
inter-arterial course



LCA from right sinus with  
retro-aortic course



Kinking of the proximal LCA  
after arterial switch procedure

## Protocol

1. **Anatomy**
2. **LV function**
3. **T1 mapping** (optional)
4. **Oedema**
5. **RV function**
6. **LGE**

## Report

1. **Dimensions** (corrected for BSA) and **function**
  - LV: EDV, ESV, SV, EF, end-diastolic diameter
  - RV: EDV, ESV, SV, EF
2. Presence and severity of **valvular regurgitation**
3. Presence, location, and extent of **fibrosis**

## Key Points

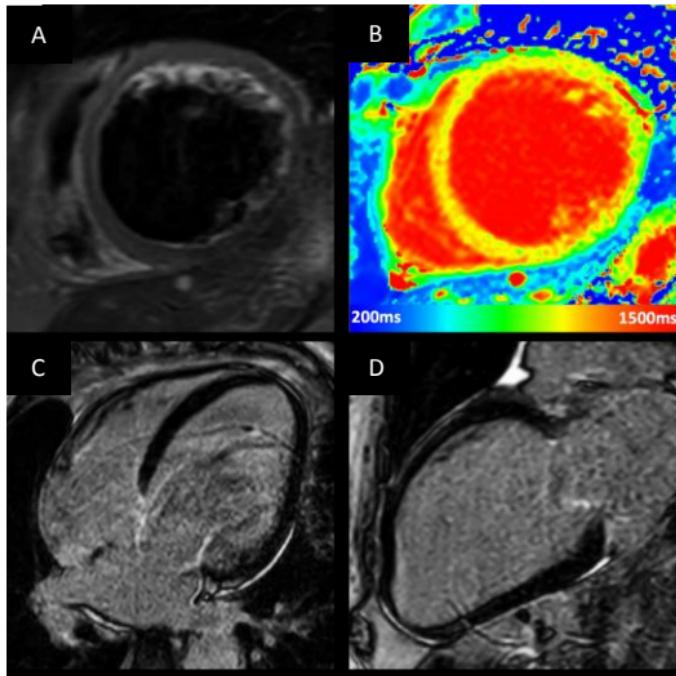
1. Mid-wall fibrosis indicative of DCM
2. **Risk factors for sudden cardiac death:**
  - LV impairment, EF <35%
  - Frequent repetitive NSVT
  - Presence and extent of mid-wall fibrosis
  - Raised ECV

## Tips & Tricks

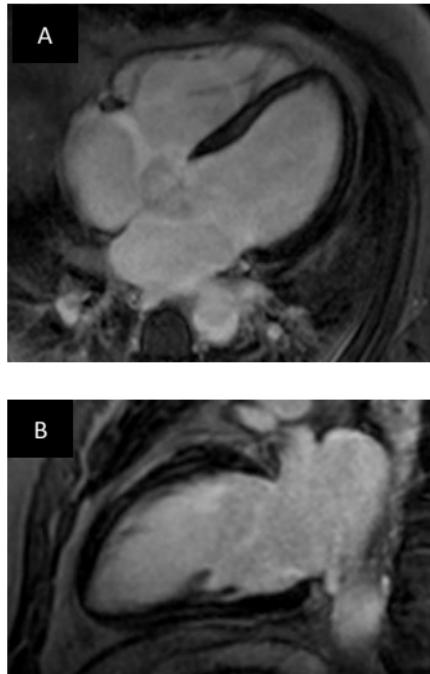
1. Use **acceleration techniques** to reduce breath-hold times
2. Consider **unrecognized CAD** if you identify:
  - Marked regional wall motion abnormalities
  - Subendocardial or transmural hyperenhancement on LGE
3. Consider abnormal vascular connections / shunts
4. **Tagging** may help identify wall motion abnormalities
5. Perfusion imaging can be difficult to interpret (thin myocardium, presence of scar and slower flow)

# Dilated Cardiomyopathy

40



**Tissue Characterisation.** T2w SA (A): no evidence of oedema; Native T1-map SA (B): slightly higher native T1 values; LGE 4CH (C) and 2CH (D): no evidence of fibrosis



**LGE 4CH (A) and 2CH (B):** typical mid-wall fibrosis, which, however is missing in most DCM patients

## Protocol

1. Anatomy
2. LV function
3. LVOT cines (2 orthogonal views)
4. Velocity encoding in- and through- LVOT planes
5. T1 mapping (optional)
6. LV tagging (3 SA slices, 4CH) – optional
7. LGE

## Report

1. Dimensions, mass (corrected for BSA) and function  
EDV, ESV, SV, EF and mass
2. Thickening and function of myocardial segments
3. Presence of LVOT obstruction at rest
4. Presence of systolic anterior motion (SAM)
5. Presence and extent of fibrosis

## Key Points

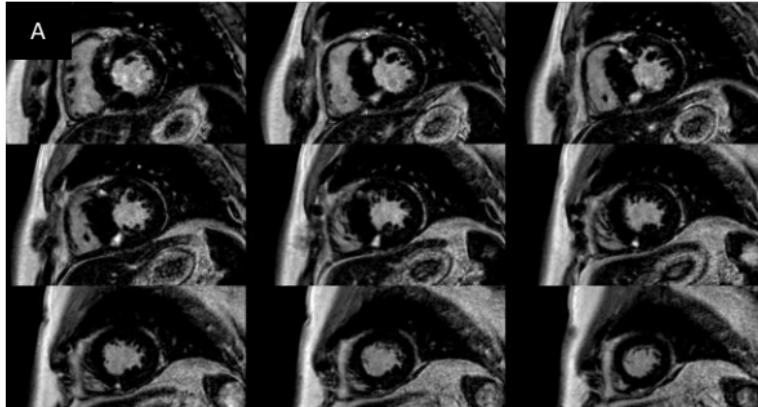
1. HCM criteria
  - Wall thickness >15mm (>13mm in familial HCM; >20mm if Afroamerican/black) in the absence of other causes
  - Septal to lateral wall thickness ratio >1.3
  - Apical to basal wall thickness ratio >=1.3 to 1.5 in apical HCM
2. Consider possible obstruction under stress conditions

## Tips & Tricks

1. LGE at the insertion points of the RV to the LV is non-specific and often seen even in normal subjects
2. Suggestive of HCM:
  - Localized hypertrophy
  - Reduced contraction of hypertrophied segments (tagging may help visualise)
  - Presence of LGE
3. Myocardial crypts may help to define HCM mutation carriers without LV hypertrophy

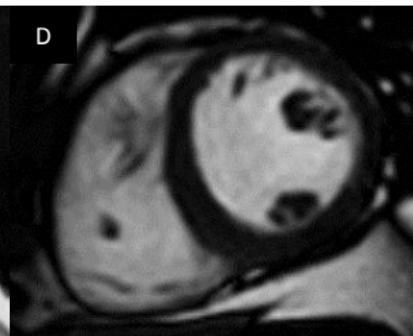
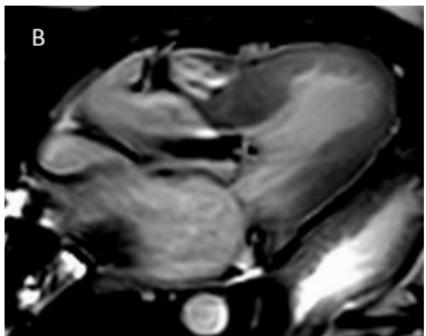
# Hypertrophic Cardiomyopathy

42



## HCM – Common findings

- **LGE SA stack (A):** severe fibrosis at the RV insertion points
- **Cine 3CH (B):** LVOT obstruction due to SAM of the mitral valve leaflets.
- **Cine 2CH (C):** multiple myocardial crypts in the inferior wall. These crypts may help to define HCM mutation carriers without LV hypertrophy. Note that myocardial crypts can be easily missed on **SA views (D)** due to partial volume



**Protocol**

1. **Anatomy**
2. **LV function (RV function)**
3. **Oedema**
4. **EGE / LGE**
5. **T1 Mapping**

**Report**

1. **Dimensions, mass** (corrected for BSA), and **function**
  - LV: EDV, ESV , SV, EF, longitudinal function, mass
  - RV: EDV, ESV, SV, EF, longitudinal function
  - Regional wall motion abnormalities
  - Thickness of interatrial septum
2. **Valve regurgitation**
3. **LGE pattern**
4. **Pericardial / pleural effusion**

**Key Points**

1. **Restrictive LV pattern** (non-dilated ventricles, preserved LV function, restrictive filling pattern, enlarged LA / RA) and global LV hypertrophy
2. **LGE**
  - On Look-Locker images the blood pool and myocardium cross zero (appear black) at similar timepoints
  - Myocardial nulling difficult to achieve on LGE images despite good technique
  - Predominantly global sub-endocardial distribution
  - Other patterns such as patchy subendocardial or transmural enhancement can also occur
3. **Atrial septal hypertrophy of >6mm** (in <20% of cases)
4. **Pericardial and pleural effusion** are common

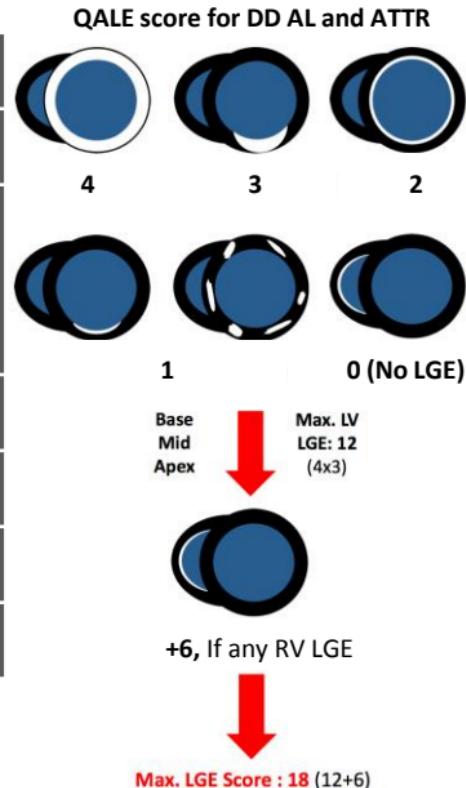
**Tips & Tricks**

1. Cross check skeletal muscle signal intensity while choosing optimal inversion time
2. For differential diagnosis AL amyloidosis vs. ATTR amyloidosis see table on next page

# Amyloidosis

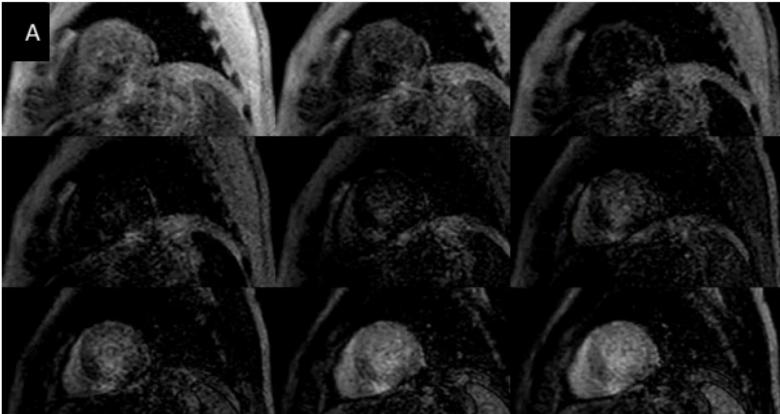
44

	AL amyloidosis	ATTR amyloidosis
LV mass	<ul style="list-style-type: none"> <li>Mildly increased <math>&lt; 100 \text{ g/m}^2</math></li> </ul>	<ul style="list-style-type: none"> <li>Markedly increased <math>&gt; 100 \text{ g/m}^2</math></li> </ul>
Septum thickness	Septum AL < Septum ATTR	
LGE	<ul style="list-style-type: none"> <li>Less extensive LGE</li> <li>Often (global) subendocardial pattern</li> <li>QALE score <math>&lt; 13</math></li> </ul>	<ul style="list-style-type: none"> <li>More extensive LGE</li> <li>Often more diffuse and transmural pattern</li> <li>QALE score <math>\geq 13</math></li> </ul>
Native T1	$> 1050 - 1150 \text{ ms}$ Native T1 <sub>AL</sub> > Native T1 <sub>ATTR</sub>	
ECV	$> 0.40$ ECV <sub>AL</sub> < ECV <sub>ATTR</sub>	
Therapy	<ul style="list-style-type: none"> <li>Chemotherapy</li> </ul>	<ul style="list-style-type: none"> <li>Novel TTR-specific treatment (Phase III)</li> </ul>
Prognosis	<ul style="list-style-type: none"> <li>Worse (despite less extensive LGE)</li> </ul>	<ul style="list-style-type: none"> <li>Better (despite more extensive LGE)</li> </ul>



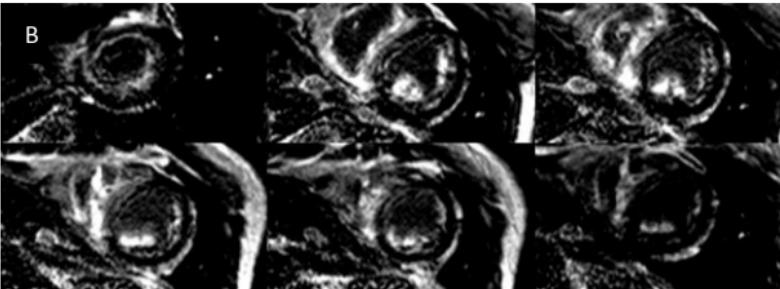
AL - light chain amyloid

#### ATTR - transthyretin-related amyloidosis

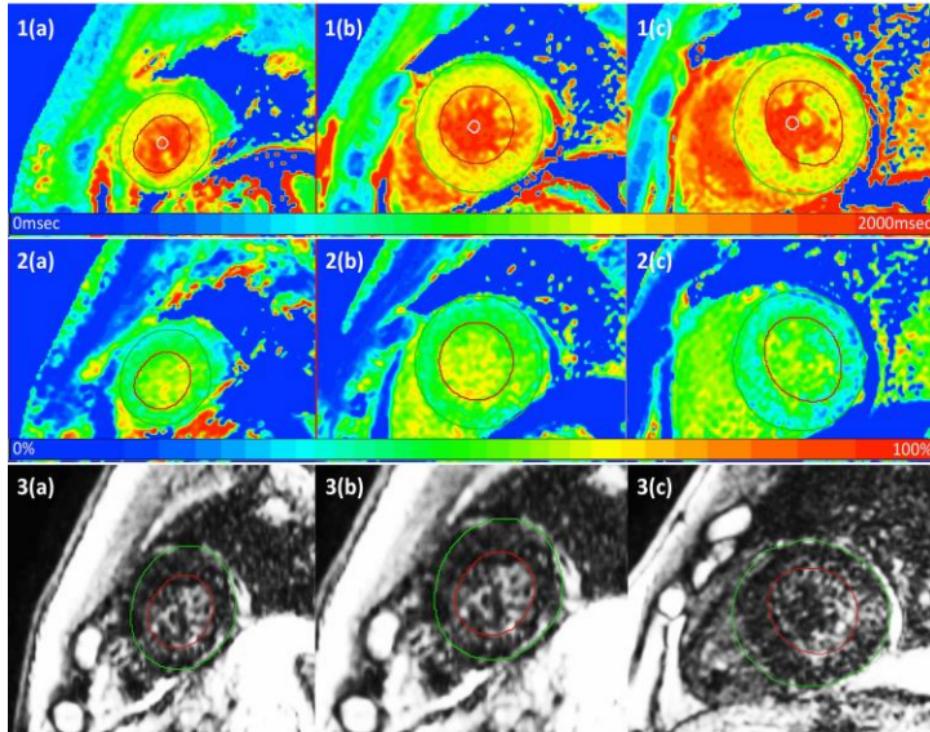


68 year old patient with suspected cardiac amyloidosis

- **Look Locker post contrast (A):** blood and myocardium are nulled at a similar TI.
- **LGE SA stack (B):** predominantly subendocardial enhancement in myocardium; note poor nulling of myocardial signal.



# Amyloidosis



**73 year old patient with suspected cardiac amyloidosis**

- Native T1 maps (1) acquired with a MOLLI method at 1.5T: diffusely elevated native T1 values ( $> 1000$  ms)
- Extra-Cellular Volume maps (2): diffusely elevated ECV values ( $>35\%$ )
- LGE SA (3): poor nulling of myocardial signal and islets of patchy enhancement.

a: apex, b: midventricular,  
c: base

**Protocol**

1. Anatomy
2. LV function
3. T1 Mapping
4. LGE

**Report**

1. Dimensions, mass (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF, longitudinal function, mass
  - Regional wall motion abnormalities
2. LGE pattern
3. Native T1 and ECV (if available)

**Key Points**

1. General
  - Multi-system lysosomal storage disease
  - X-linked (men > women)
2. Disease pathophysiology:
  - Accumulation of globotriaosylceramide in the vascular endothelium, leading to ischaemia and infarction in the kidney, heart and brain

**3. Concentric LVH**

- Relatively late disease manifestation (3rd decade in men, 4th decade in women)
- Patterns of hypertrophy often indistinguishable from HCM
- LVH associated with progressive myocardial fibrosis

**4. LGE**

- Present in 50% of cases
- Mid-wall or subepicardial LGE of the inferolateral wall of mid to basal LV

**5. T1 mapping**

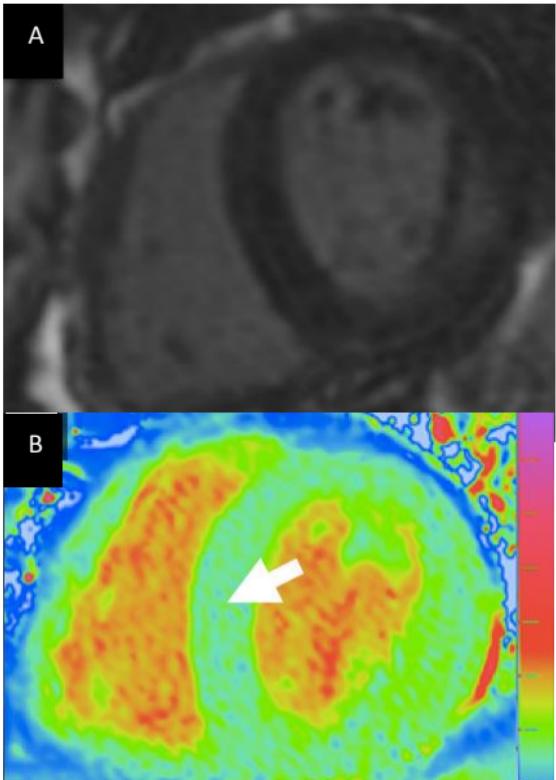
- Native T1↓ of septum (< 940ms (1.5T))
- ECV↓
- Inferolateral LGE: pseudonormalisation native T1 normal/↑ possible if the effects of replacement fibrosis exceed the fatty-related T1 decrease

**Tips & Tricks**

- AFD is underdiagnosed (prevalent in dialysis patients, late-onset "HCM")

# Anderson Fabry Disease

48



## 34 year old patient with Anderson Fabry Disease

- **LGE SA (A):** hyperenhancement of basal inferolateral and inferior segments
- **Native T1 map (B):** low septal T1 (780 ms) appearing blue (white arrow).

**Protocol**

1. Anatomy and LV/RV function
2. T2\*

**Report**

1. Dimensions, mass (corrected for BSA), and function
  - LV: EDV, ESV, SV, EF, longitudinal function, mass
  - RV: EDV, ESV, SV, EF, longitudinal function
2. T2\* (ms), R2\* (Hz) and dry weight iron content

**Key Points**

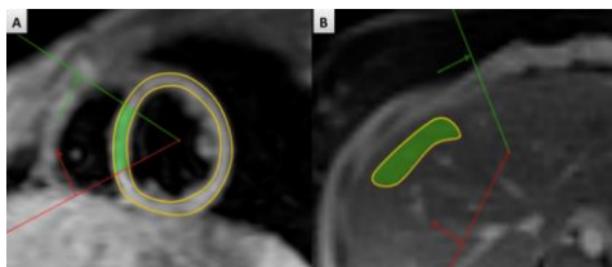
1. Iron overload cardiomyopathy
  - Potentially reversible cause of heart failure under effective therapy
  - **Dilated phenotype** – majority of patients, impaired systolic function
  - **Restrictive phenotype** - non-dilated ventricles, preserved systolic function, diastolic dysfunction, enlarged atria
2. Risk of developing heart failure
  - T2\* > 20 ms: low
  - T2\* 10-20 ms: intermediate
  - T2\* <10 ms: high

**3. Other diagnostic signs**

- Focal signal loss in native T1- and T2-weighted images
  - Abnormally "dark" liver
4. Serial follow-up of iron loading to guide chelation therapy
  5. Isolated cardiac or liver involvement is possible

**Tips & Tricks**

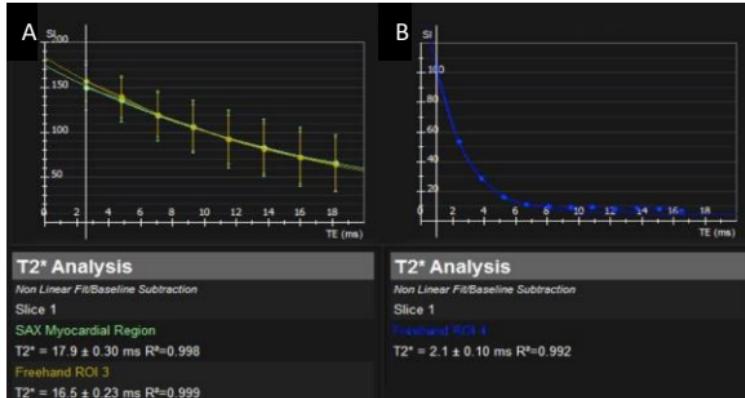
1. Assess T2\* values in the septum (fewer artefacts) as iron deposition is similar in all LV segments
2. High correlation of T2\* values and native T1 values ( $T2^*\downarrow$ , native T1 $\downarrow$ )



T2\* contours: ROIs are placed in the ventricular septum (A) and the liver (B).

# Iron Overload Cardiomyopathy

50



**T2\* calculation:** Graphs of the relaxation of T2\* values over time to compute T2\* values of **myocardium (A)** and **liver (B)**. T2\* of the ventricular septum was 16.5ms consistent with mild myocardial iron overload and intermediate risk of developing heart failure, T2\* of liver was 2.1ms consistent with severe iron overload of the liver.

Myocardial T2*(ms)	Myocardial R2*(Hz)	Dry weight (MIC) (mg/g)	Hepatic T2* (ms)	Hepatic R2* (Hz)	Dry weight (LIC) (mg/g)	Classification
≥20	≤ 50	≤ 1.16	≥15.4	≤ 65	≤ 2	None
14-20	50 – 71	1.16 – 1.8	4.5-15.4	65 - 224	2-7	Mild
10-14	71 – 100	1.8 – 2.7	2.1 – 4.5	224 - 475	7-15	Moderate
≤10	≥ 100	≥ 2.7	≤2.1	≥ 475	≥ 15	Severe

Liver Iron Content (LIC) calculated according to Ref 10; Myocardial Iron Content (MIC) calculated according to Ref 11

## Protocol

1. **Anatomy**
2. **LV function (RV function)**
3. **Oedema**
4. **LGE**

## Report

1. **Dimensions, mass** (corrected for BSA), and **function**
  - LV: EDV, ESV, SV, EF, longitudinal function, mass
  - RV: EDV, ESV, SV, EF, longitudinal function
  - **Regional wall motion abnormalities**
2. **Myocardial granulomas** on LGE images
3. **Extra-cardiac findings**

## Key Points

1. **Restrictive LV pattern** (non-dilated ventricles, preserved LV function, restrictive filling pattern, enlarged LA / RA)
2. **Cardiac involvement:**
  - in about 25% of patients with systemic sarcoidosis

## 3. Myocardial granulomas on LGE images:

- Intramural
- Spotty
- Predominantly basal lateral
- Respond to immunosuppressive drugs
- Enhancement not in CAD territory distribution

## 4. LV dysfunction is common

## 5. Focal oedema indicates inflammation

- may mimic hypertrophic cardiomyopathy

## 6. Usually accompanied with extra-cardiac findings:

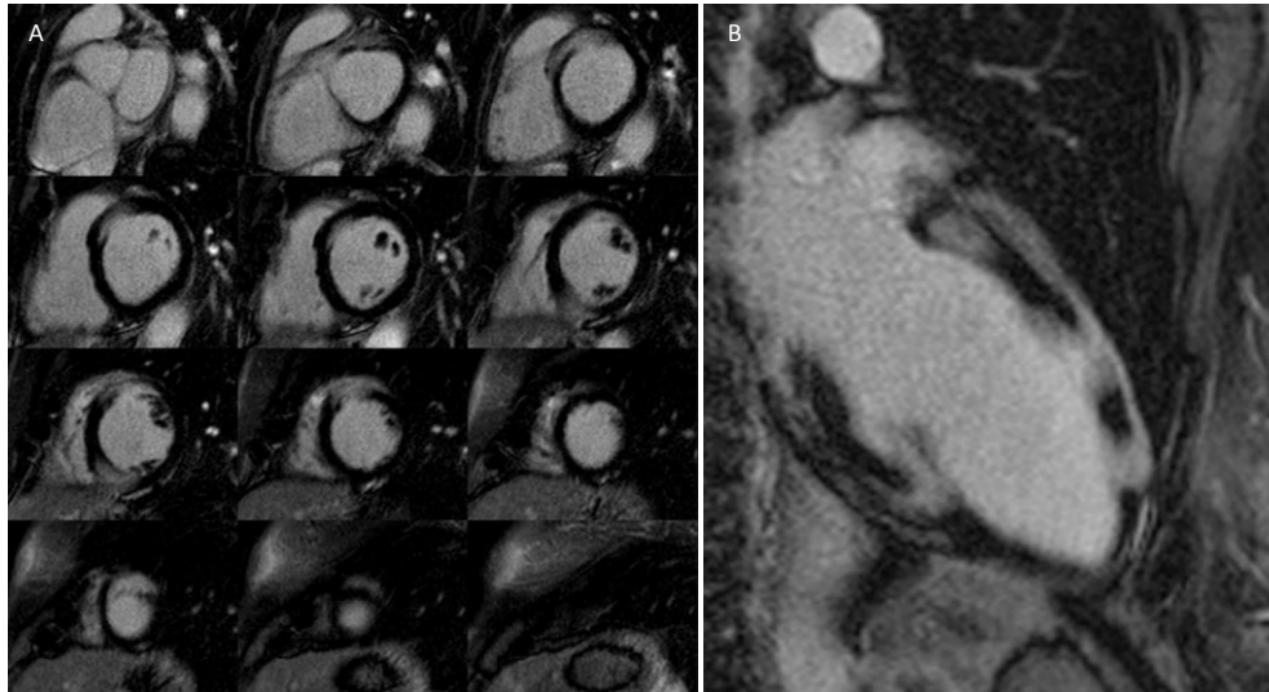
- Hilar lymphadenopathy
- Involvement of any other organ system possible

## Tips & Tricks

1. High degree AV nodal blocks, AF and NSVT are common in sarcoidosis

# Sarcoidosis

52



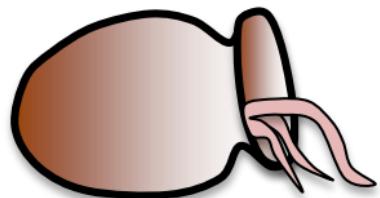
**LGE SA stack (A) and 2CH (B):** Typical spotty enhancement in the regions of wall motion abnormalities representing granulomas. Note, that the enhancement is not in a coronary artery territory distribution.

## Protocol

1. Anatomy
2. LV function (RV function)
3. Oedema
4. LGE
5. T2 mapping for oedema detection (if available)

## Report

1. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF
  - RV: EDV, ESV, SV, EF
  - Regional wall motion abnormalities
2. Presence of oedema
3. Presence of LGE

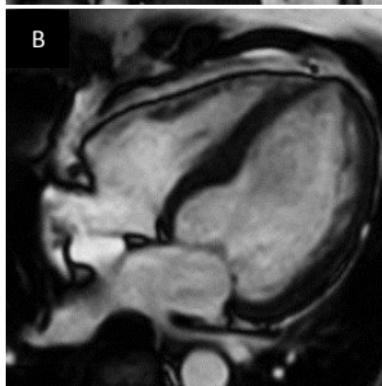
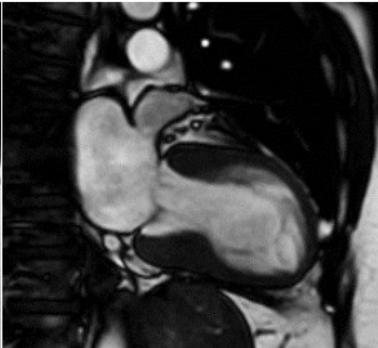
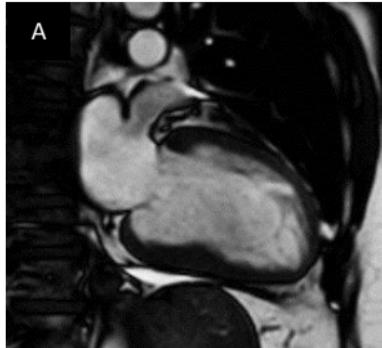


## Key Points

1. Transient acute left ventricular dysfunction due to neurogenic myocardial stunning
2. Usually in **post-menopausal women** and in the setting of **acute emotional or physical stress**
3. Recovery takes place over a few days with full recovery over a few weeks
4. **Typical Tako-Tsubo pattern**
  - Apical akinesia / ballooning
  - Basal / mid-ventricular hyperkinesia
5. **Inverted Tako-Tsubo pattern**
  - Mid-ventricular and basal akinesia / ballooning
  - Apical hyperkinesia
6. Oedema in the areas of wall motion abnormalities
7. Classically **NO** signs of LGE
  - Infarct-like hyperenhancement has been described in a few rare cases

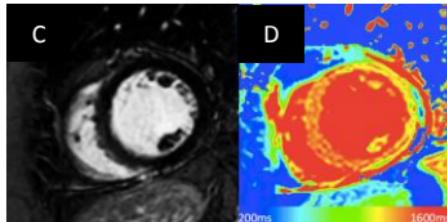
# Tako-Tsubo Cardiomyopathy

54



55-year old lady with acute onset chest pain and troponin rise. Normal invasive coronary angiography. Recent bereavement in immediate family.

- **Cine 2CH (A) and 4CH (B) diastolic and systolic frames:** classical apical ballooning with preserved basal contraction
- **LGE SA (C):** no evidence of myocardial infarction.
- **Native T1 map SA (D):** significantly raised T1 values in mid ventricle suggestive of myocardial oedema



# Left Ventricular Non - Compaction Cardiomyopathy

## Protocol

1. Anatomy
2. LV function
3. LGE

## Report

1. Dimensions, mass (corrected for BSA) and function
  - LV: EDV, ESV , SV, EF
  - Do not routinely report the extent of trabeculation
2. Overall cardiac appearance: “ otherwise normal” vs. “structural or developmental” abnormality
3. Pre-test probability for LVNC: family history, neuromuscular disorder, significant arrhythmia

## Key Points

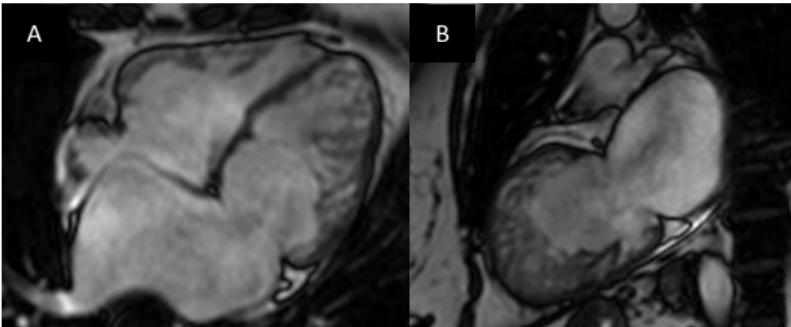
1. Current diagnostic criteria:
  - Multiple criteria (\* table)
  - No need for routine reporting of the trabeculation
2. Excessive trabeculation is benign and not associated with adverse outcomes
3. Diagnosis of the LVNC cannot be based on imaging criteria alone
  - Pre-test probability is relevant
  - Often over-diagnosed, particular in DCM (thin compacted myocardium) and in patients of Afro-American descent
  - Significant if found with other morphological/developmental abnormalities, neuromuscular disorders and/or family history of cardiomyopathy

# Left Ventricular Non-Compaction: Criteria

56

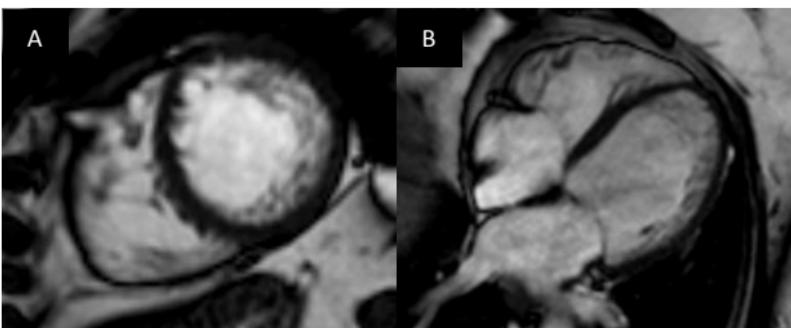
Modality	Echo (ref 43)	CMR (ref 44)	CMR (ref 45)	CMR (ref 46)	CMR (ref 47)
<b>Criteria</b>	<ul style="list-style-type: none"> <li>• NC/C in parasternal SA</li> <li>• Deep perfused inter-trabecular recesses on Colour Doppler</li> <li>• Decreased thickening and hypokinesia within, but not limited to, NC segments</li> </ul>	<ul style="list-style-type: none"> <li>• Two-layered myocardium</li> <li>• Long axis SSFP cine</li> <li>• Measured at the most pronounced trabeculation</li> <li>• Measurement perpendicular to compacted myocardium</li> </ul>	<ul style="list-style-type: none"> <li>• Total LV mass (SA SSFP cines)</li> <li>• Compacted myocardial mass (SA SSFP cines)</li> <li>• Trabecular mass = Total LV mass minus compacted myocardial mass</li> </ul>	<ul style="list-style-type: none"> <li>• Apical SA views 16 to 24 mm from the true apical slice</li> <li>• Region with the largest NC/C ratio</li> </ul>	<ul style="list-style-type: none"> <li>• SA views</li> <li>• Loss of base-to-apex fractal dimension (FD)* gradient</li> </ul> <p>*FD is an index of how completely an object fills space.</p>
<b>Cardiac phase</b>	End-systole	End-diastole	End-diastole	End-systole	End-diastole
<b>Definition</b>	NC/C ratio > 2	NC/C ratio > 2.3	Trabecular mass > 20%	NC/C ratio ≥ 2	FD ≥ 1.30
<b>Sensitivity specificity</b>	n/a	Sens 86% Specif 99%	Sens 93.7% Specif 93.7%	n/a	Sens 83-100% Specif 86-100%

# Left Ventricular Non-Compaction Cardiomyopathy



## Patient with family history of cardiomyopathy

- **Cine 4CH (A) and 2CH (B):** marked trabeculation of all LV walls except the interventricular septum
- Excessive LV trabeculation is a part of gross morphological abnormalities and positive family history
- This is most likely LV non-compaction cardiomyopathy



## Patient with no clinical features of LVNC

- **Cine SA (A) and 4CH (B):** marked LV trabeculation, primarily in the lateral wall
- No other abnormalities
- Most likely anatomical variant
- Consider that the diagnosis of LVNC is challenging and may not be based on imaging criteria alone

# Arrhythmogenic Right Ventricular Cardiomyopathy

58

## Protocol

1. **Anatomy**
2. **LV function**
3. **RV function** (transaxial and RVOT)
  - **Slice thickness 6-8mm without inter-slice gap**
4. **T1w axial black blood** (optional)
5. **T1w axial fat suppressed black blood** (optional)
6. **LGE** in same orientations
  - **T1 nulling for RV**

## Report

1. **Dimensions, mass** (corrected for BSA) and **function**
  - LV: EDV, ESV, SV, EF, longitudinal function, mass
  - RV: EDV, ESV, SV, EF, longitudinal function
    - **RV regional wall motion abnormalities** (inflow, apex, outflow)
2. Presence of **morphological RV abnormalities** (**aneurysms, outpouchings**)
3. Presence of **fatty RV or LV infiltration**
4. Presence and extent of **fibrosis**

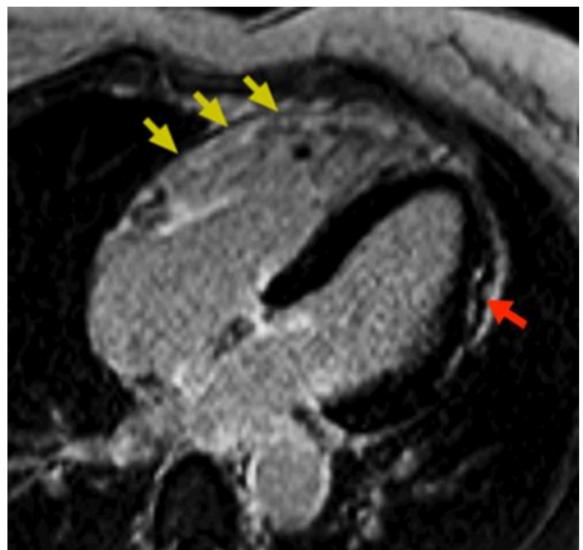
## Key Points

1. **Diagnosis cannot be based on imaging criteria alone**
  - See modified 2010 Task Force ARVC criteria
2. **RV wall motion abnormalities at the moderator band insertion point is common in normal subjects**

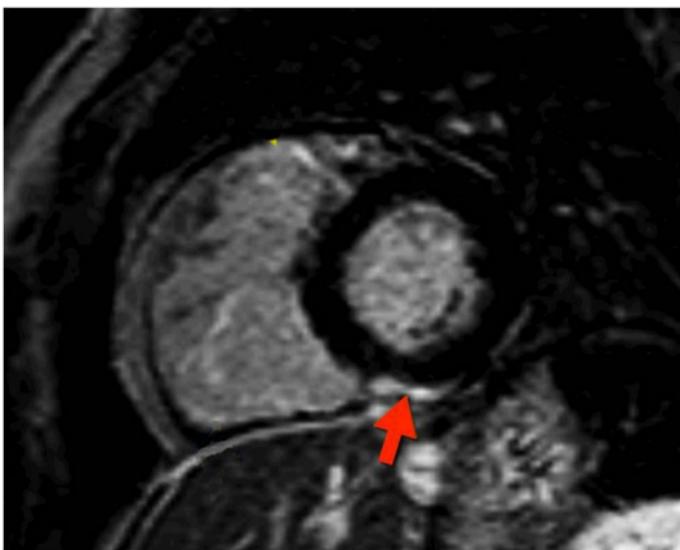
## Tips & Tricks

1. **Focus on RV volumes and functional RV abnormalities**
2. Consider antiarrhythmic drugs in patients with VES
3. Consider alternative causes (abnormal vascular connections / shunts) in patients with dilated RV

# Arrhythmogenic Right Ventricular Cardiomyopathy



**LGE 4CH:** Fibrosis in the RV free wall (yellow arrows) with involvement of the LV lateral wall (red arrow) in a non-ischaemic pattern.



**LGE SA:** Fibrosis in the LV (red arrow) in a non-ischaemic pattern.

# ARVC diagnostic criteria I

## 1. Global or regional dysfunction and structural alterations

- Major**
- Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
  - and 1 of the following:
    - Ratio of RV end-diastolic volume to BSA  $\geq 110\text{mL/m}^2$  (male) or  $\geq 100\text{mL/m}^2$  (female)
    - or RV ejection fraction  $\leq 40\%$
- Minor**
- Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
  - and 1 of the following:
    - Ratio of RV end-diastolic volume to BSA  $\geq 100$  to  $< 110\text{mL/m}^2$  (male) or  $\geq 90$  to  $< 100\text{mL/m}^2$  (female)
    - or RV ejection fraction  $> 40\%$  to  $\leq 45\%$

### Definite diagnosis

- 2 major or 1 major and 2 minor criteria
- 4 minor criteria

### Borderline diagnosis

- 1 major and 1 minor
- 3 minor criteria

### Possible diagnosis

- 1 major or 2 minor criteria

## 2. Tissue characterization of wall (Histological)

- Major**
- Residual myocytes  $< 60\%$  by morphometric analysis (or  $< 50\%$  if estimated), with fibrous replacement of the RV free wall myocardium in  $\geq 1$  sample, with or without fatty replacement of tissue on endomyocardial biopsy
- Minor**
- Residual myocytes 60% to 75% by morphometric analysis (or 50% to 65% if estimated), with fibrous replacement of the RV free wall myocardium in  $\geq 1$  sample, with or without fatty replacement of tissue on endomyocardial biopsy

## 3. Repolarization abnormalities

- Major**
- Inverted T waves in right precordial leads (V1, V2, and V3) or beyond in individuals  $> 14$  years of age (in the absence of complete right bundle-branch block QRS  $\geq 120\text{ms}$ )
- Minor**
- Inverted T waves in leads V1 and V2 in individuals  $> 14$  years of age (in the absence of complete right bundle-branch block) or in V4, V5, or V6
  - Inverted T waves in leads V1, V2, V3, and V4 in individuals  $> 14$  years of age in the presence of complete right bundle-branch block

**4. Depolarization / conduction abnormalities**

- Major**
- Epsilon wave (reproducible low-amplitude signals between end of QRS complex to onset of the T wave) in the right precordial leads (V1 to V3)
- Minor**
- Late potentials by SAECG in  $\geq 1$  of 3 parameters in the absence of a QRS duration of  $\geq 110$ ms on the standard ECG
  - Filtered QRS duration (fQRS)  $\geq 114$ ms
  - Duration of terminal QRS  $< 40 \mu\text{V}$  (low-amplitude signal duration)  $\geq 38$ ms
  - Root-mean-square voltage of terminal 40ms  $\leq 20 \mu\text{V}$
  - Terminal activation duration of QRS  $\geq 55$ ms measured from the nadir of the S wave to the end of the QRS, including R', in V1, V2, or V3, in the absence of complete right bundle-branch block

**5. Arrhythmias**

- Major**
- Nonsustained or sustained ventricular tachycardia of left bundle-branch morphology with superior axis (negative or indeterminate QRS in leads II, III, and aVF and positive in lead aVL)
- Minor**
- Nonsustained or sustained ventricular tachycardia of RV outflow configuration, left bundle-branch block morphology with inferior axis (positive QRS in leads II, III, and aVF and negative in lead aVL) or of unknown axis  $> 500$  ventricular extrasystoles per 24 hours (Holter)

**6. Family history**

- Major**
- ARVC confirmed in a first-degree relative who meets current Task Force criteria
  - ARVC confirmed pathologically at autopsy or surgery in a first-degree relative
  - Identification of a pathogenic mutation<sup>†</sup> categorized as associated or probably associated with ARVC in the patient under evaluation
- Minor**
- History of ARVC in a first-degree relative in whom it is not possible or practical to determine whether the family member meets current Task Force criteria
  - Premature sudden death ( $< 35$  years of age) due to suspected ARVC in a first-degree relative
  - ARVC confirmed pathologically or by current Task Force Criteria in second-degree relative

# Becker and Duchenne Muscular Dystrophies

62

## Protocol

1. Anatomy
2. LV/RV function
3. T1 mapping (native & post-contrast)
4. LGE

## Report

1. Dimensions, mass (corrected for BSA) and function
2. LV: EDV, ESV, SV, EF, longitudinal function
3. Regional wall motion abnormalities
4. LGE pattern
5. Native T1 and ECV (if available)

## Key Points

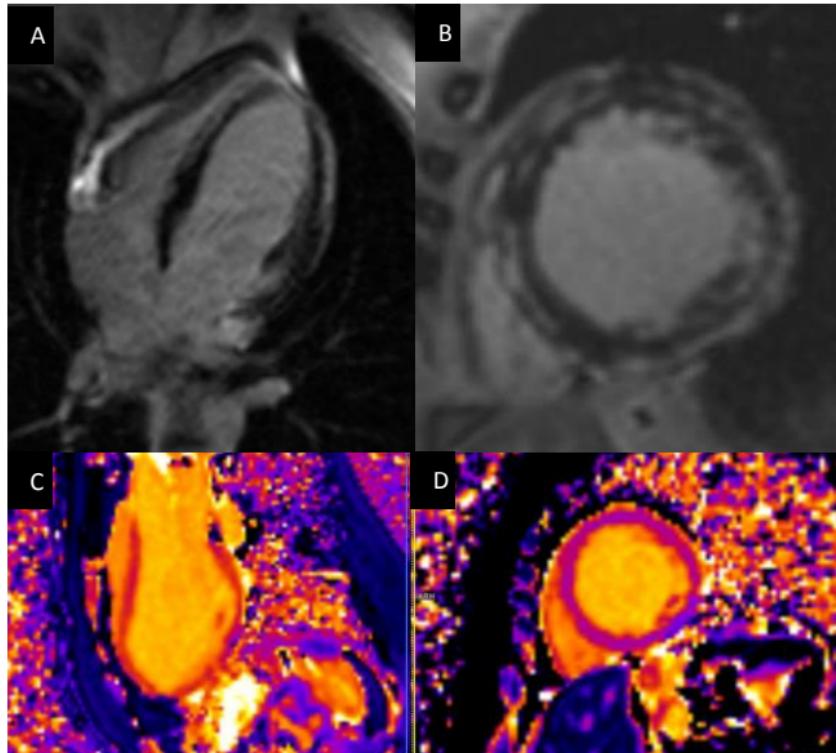
1. BMD and DMD commonest muscular dystrophies (80% of all muscular dystrophies) with high rate of cardiomyopathy
2. X-linked (men > women); female carriers may also present cardiac involvement and similar pattern of myocardial fibrosis
3. Disease pathophysiology: affecting the synthesis of dystrophin
4. ECG: increased R-to-S ratio in the right precordial leads, deep lateral Q waves, conduction abnormalities, arrhythmias (SVT>VT)

5. Biventricular impairment, hypertrophy and/or dilatation
6. LGE: subepicardial fibrosis inferolateral (BMD, DMD) remarkably similar to viral myocarditis
7. Fat-suppressed imaging/native T1 mapping may help delineate the extent of myocardial fibrosis/myocardial fat infiltration
8. T1 mapping
  - ECV elevated (29+/-6%)
  - shorter T1 post-contrast than in controls may detect diffuse fibrosis
  - pseudonormalisation of native T1 if effects of replacement fibrosis cancel out fat related T1 decrease

## Tips & Tricks

1. Cardiac involvement characterized by myocardial fibrosis - DCM, heart failure, (malignant) arrhythmias, heart block
2. LGE can be "transmural" in the absence of coronary artery disease (additive prognostic value)
3. Early detection for early heart failure therapy may delay the progression of LV dysfunction
4. Clinically overt heart failure in dystrophinopathies may be delayed or absent, due to relative physical inactivity

# Becker and Duchenne Muscular Dystrophy



**25 year old man with Duchenne Muscular Dystrophy**

- **LGE 4CH (A) and SA (B):** septal mid-wall enhancement and patchy inferolateral enhancement (fibro-fatty replacement)
- **Native T1 map 2CH (C) and SA (D):** decreased native T1 values in the mid inferolateral wall indicating fatty infiltration

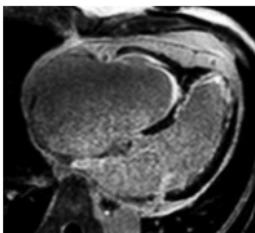
# Endomyocardial Fibrosis

## Protocol

1. Anatomy
2. LV function (RV function)
3. Oedema
4. EGE / LGE

## Report

1. Dimensions, mass (corrected for BSA), and function
  - LV: EDV, ESV, SV, EF, longitudinal function, mass
  - RV: EDV, ESV, SV, EF, longitudinal function
2. Presence and extent of fibrosis
3. Presence of ventricular thrombus



**LGE 4CH:** endomyocardial pattern of fibrosis in both ventricles at the apex ("V sign"). Severely dilated atria

## Key Points

1. Tropical or non-tropical (Löffler's syndrome/ eosinophilic cardiomyopathy) eosinophilic endomyocardial fibrosis
2. Usually increased eosinophil count
3. Restrictive LV pattern (non-dilated ventricles, preserved LV function, restrictive filling pattern, enlarged LA / RA)
4. Endocardial thickening and scarring
5. RV involvement in about 50% of cases
6. Ventricular thrombi are common (EGE images)
7. LGE pattern
  - Circumferential sub-endocardial hyperenhancement
  - Rarely affects more than 50% of the wall thickness
  - Endomyocardial pattern of fibrosis in both ventricles at the apex ("V sign").

## Tips & Tricks

1. Hypereosinophilia and cardiac involvement are also seen in other diseases, i.e. Churg–Strauss syndrome

**Protocol**

1. **Anatomy**
2. **LV function** (RV function)
3. **T1 mapping** (optional)
4. **T2 mapping** (optional)
5. **Oedema**
6. **LGE**

**Report**

1. **Dimensions** (corrected for BSA) and **function**
  - LV: EDV, ESV, SV, EF
  - RV: EDV, ESV, SV, EF
  - **Regional wall motion abnormalities**
2. Presence and location of **oedema**
3. Presence and location of **LGE**
4. **Pericardial effusion / enhancement**

**Key Points**

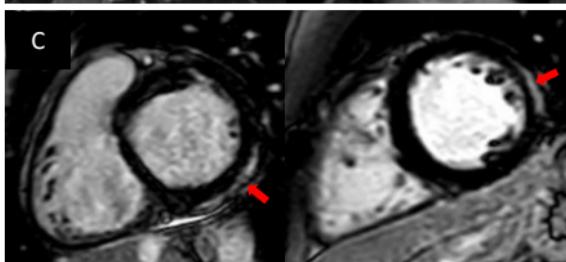
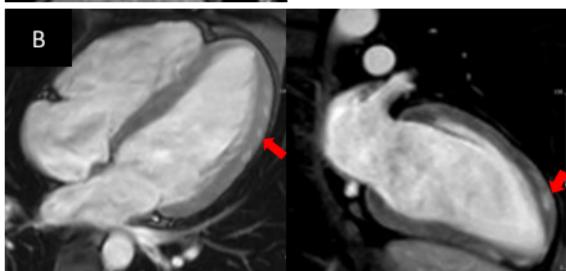
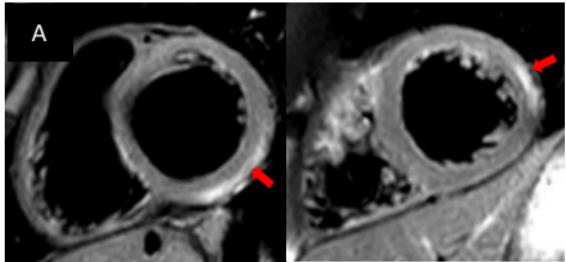
1. **Diagnostic CMR criteria**  
**Myocardial inflammation ( $\geq 2$  of the following criteria)**  
**Myocyte injury and / or scar (if focal lesion is present)**

- **Regional or global myocardial SI increase on T2w**  
SI ratio of myocardium over skeletal muscle of  $\geq 2.0$
  - **Global myocardial SI increase on EGE**  
SI ratio of myocardium over skeletal muscle of  $\geq 4.0$  or absolute myocardial enhancement of  $\geq 45\%$
  - **At least 1 focal lesion with non-ischaemic regional distribution (sub-epicardial layer or mid-wall)**  
Infarction always involves sub-endocardial layer
2. Presence of **LV dysfunction** or **pericardial effusion** provides additional, supportive evidence
  3. **Repeat scan** in 1-2 weeks after the first study, if
    - **None of the criteria** are present plus **very recent onset of symptoms** plus **strong clinical evidence**
    - **One of the criteria** is present

**Tips & Tricks**

1. **Right ventricular dysfunction** seems to be the greatest predictor of mortality and cardiac transplantation

# Myocarditis



**23 year old patient with chest pain and raised troponin**

- **T2w STIR SA (A):** increased subepicardial signal of the basal infero-lateral segment and mid to apical lateral wall, suggesting myocardial oedema in these areas
- **EGE 4CH and 2CH (B):** patchy hyperenhancement of the mid-apical lateral wall and apical anterior wall
- **LGE SA (C), 4CH (D) and 2CH (E):** subepicardial hyperenhancement in the basal inferior and infero-lateral walls and mid lateral and apical anterior and anterolateral walls.

**Protocol**

1. Anatomy
2. LV function (RV function)
3. Oedema
4. LGE

**Report**

1. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF
  - RV: EDV, ESV, SV, EF
  - Regional wall motion abnormalities
2. Presence and location of oedema
3. Presence and location of LGE
4. Pericardial effusion / enhancement



**LGE 4CH (A), 3CH (B) and SA (C):** Pericardial hyperenhancement secondary to pericardial inflammation without any pericardial effusion

# Constrictive Pericarditis

## Protocol

1. **Anatomy** including T1w and T2w
2. **LV / RV function**
3. **Real-time dynamic respiratory cine**
4. **LGE**

## Report

1. **Dimensions** (corrected for BSA) and **function**
  - LV: EDV, ESV , SV, EF
  - RV: EDV, ESV, SV, EF
2. Septal motion during normal and dynamic respiration
3. **Pericardial thickening  $\geq 3\text{mm}$**
4. Presence or absence of **RV diastolic collapse**
5. **LGE** enhancement in RV, LV and pericardium

## Key Points

1. Pericardial **thickening, calcification, scarring** with **preserved LV function**, but **impaired diastolic filling**
2. Constrictive pericarditis is usually a **chronic disease**, but consider transient constriction in inflammation states

## 3. Typical findings:

- Septal shift towards LV during inspiration
- Dilated atria
- Definitive diagnosis requires additional studies

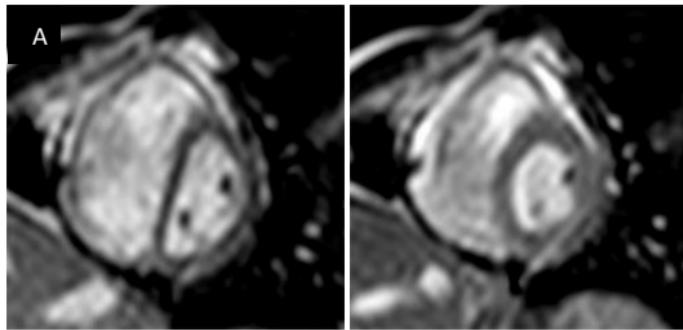
4. Constriction can be **localized** but often leads to an **impairment of biventricular filling**
5. **Common causes:** post cardiac surgery / trauma, irradiation, inflammation, connective tissue disease, idiopathic

## Tips & Tricks

1. Pericardial constriction may be present even with a normal pericardial thickness or patchy thickening
2. Real-time dynamic respiratory sequence in several SA views and in a 4-ch view (paradoxical septal motion is often being limited to one part of the septum)
3. CMR cannot conclusively detect calcification

# Constrictive Pericarditis

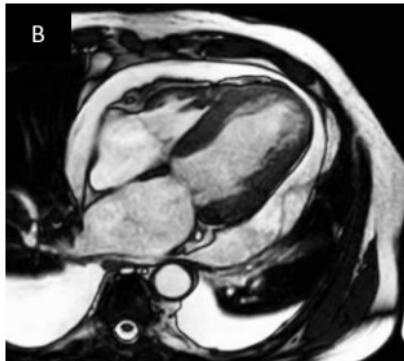
Inspiration



Expiration

**59-year-old man with history of predominantly right heart failure**

- **Real time SA (A)** showing **ventricular interdependence**: septal shift to the LV during inspiration in diastole. During expiration diastolic bulging of the interventricular septum towards the RV
- **LGE 4CH (B)**: severely thickened and enhanced pericardium. Note the moderate bi-atrial dilatation and pleural effusions.



# Pericardial Effusion

## Protocol

1. Anatomy including T1 and T2 weighted imaging
2. LV function (RV function)
3. Consider:
  - Tumour protocol
  - Valve protocol
  - Real-time free-breathing cine (2 planes)
4. LGE

## Report

1. Pericardial thickness (normal <3mm)
2. Presence and extent of pericardial effusion
3. Dimensions (corrected for BSA) and function
  - LV: EDV, ESV, SV, EF
  - Regional wall motion abnormalities
  - Septal wall motion during normal respiration and breath holding
4. Presence or absence of atrial or ventricular diastolic collapse
5. LGE in RV, LV and pericardium

## Key Points

1. Pericardial tamponade is a clinical diagnosis
  - Even a small and focal effusion can be haemodynamically significant
2. Signs of tamponade:
  - RA / LA collapse, RV / LV collapse
  - Septal shift towards LV during inspiration
3. Typical causes of pericardial effusion:
  - Global: uremic, infectious, myxoedema, neoplastic
  - Regional: postoperative, trauma, purulent, cyst

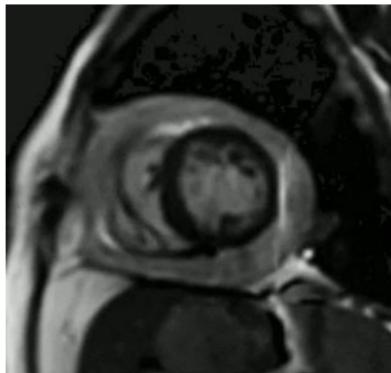
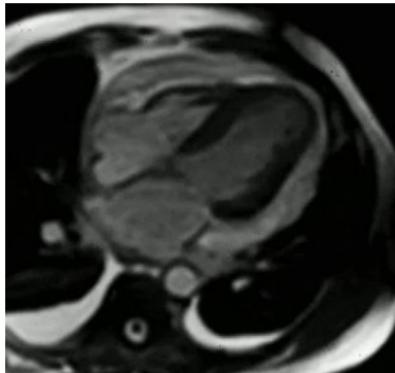
## Tips & Tricks

- Pericardial effusion and pleural effusion are both seen as high signal in cine images, but differ on TSE sequences

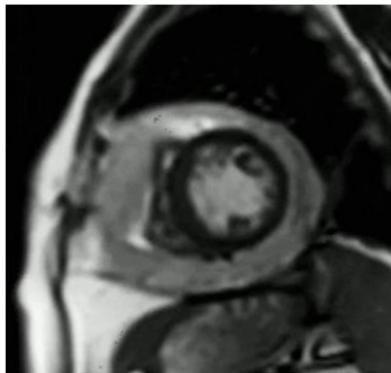
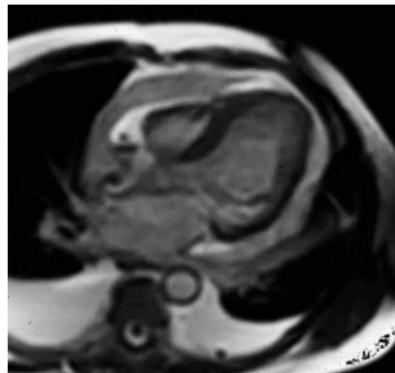
CMR appearance T1	Cine	SI (b-SSFP)
Transudate	↓	simple
Exudate	↓↑	complex
Hemorrhage	↓↑	complex
Chylous	↑↑	simple

# Pericardial Effusion

Inspiration



Expiration



**Severe Dressler syndrome in a 65-year-old patient with history of anterior STEMI**

- Real Time cine SSFP 4CH and SA: RA and RV collapse during expiration when intra-thoracic pressures rise above RA/RV pressures.
- Dilated LV with impaired systolic function and akinetic LAD territory. Large pericardial effusion and bilateral pleural effusion

# Valvular heart disease

## General

- CMR study for evaluating valvular heart disease has the following objectives:**
  - Provide insight into the mechanism of the valvular lesion (via anatomic assessment)
  - Quantify the severity of the valvular lesion
  - Discern the consequences of the valvular lesion
- Comprehensive valve assessment:**
  - LV/RV dimensions, mass, fibrosis, and function
  - Forward and regurgitant flow / fraction
  - Mean / peak velocity
  - Jet detection, direction and origin
  - Valve area by direct planimetry

Pulse Sequence	Indication
SSFP cine	<ul style="list-style-type: none"> <li>Anatomy and motion</li> <li>LV/RV volumes and function</li> </ul>
Gradient echo cine	<ul style="list-style-type: none"> <li>Valve leaflet motion</li> <li>Turbulent flow</li> </ul>
Flow velocity encoding	<ul style="list-style-type: none"> <li>Forward / regurgitant volume</li> </ul>

## Tips & Tricks

- Reduce slice thickness to <6mm
- Consider overlapping of slices

## Limitations

- Degree of stenosis or regurgitation – cine imaging**
  - Visual assessment from cine images alone is NOT recommended due to a signal void in turbulent flow
- Valve area – planimetry**
  - Correct imaging planes at the tip of the leaflets are fundamental
  - Note that a perfect 2D image plane of a 3D structure is impossible
- Flow velocity encoding– forward flow / peak velocity**
  - VENC tends to underestimate velocities due to partial volume averaging / slice orientation NOT perpendicular to the flow
- Flow velocity encoding– regurgitation volume / fraction**
  - Consider volume shift through moving aorta or PA during cardiac cycle
  - Consider regular back-flow into the coronary arteries

**Protocol**

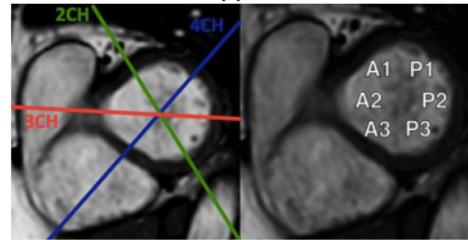
- Anatomy / LV function / RV function**
- Optimized cine views for MV:**
  - Slice thickness 5mm - consider overlapping
  - Standard long-axis views - 4CH, 2CH, 3CH
  - 3CH cine stack (perpendicular to the commissure)
- Phase contrast velocity encoded – AV (MV - unreliable)**

**Anatomy**

- MV components**
  - Leaflets (anterior and posterior)
  - Anterolateral and posteromedial commissure
  - Annulus (oval and saddle shape)
  - Sub-valvular apparatus (chordae and papillary muscles)
- Mitral valve leaflets**
  - Anterior leaflet: 3 scallops A1, A2, and A3 (tongue shaped)
  - Posterior leaflet: 3 scallops P1, P2, and P3 (crescent-shaped)
  - A1 / P1 located near to the LAA (anterolateral)

**Report**

- Dimensions, mass (corrected for BSA) and function**
  - LV and RV: EDV, ESV, SV, EF
- Mitral valve morphology**
  - Leaflets:** thickened, restricted, tethered, bowing, prolapse, or flail
  - Annulus:** thickened, fibrosis on LGE
  - Sub-valvular apparatus:** thickened, restrictive, rupture
- Mitral regurgitation**
  - MV regurgitation volume / fraction (RegV/RegF)
- Mitral stenosis**
  - MV orifice area planimetry ( $\text{cm}^2$ )
  - MV pressure gradients unreliable because of motion of MV apparatus



Mitral valve segmentation

# Mitral regurgitation

## Degenerative (primary) mitral regurgitation

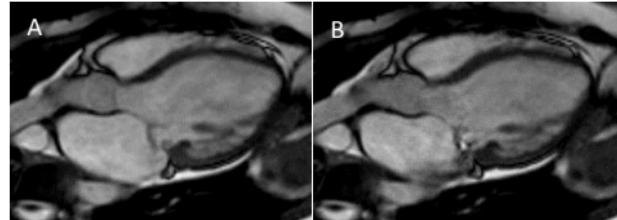
Caused by a primary abnormality of components of the valve apparatus

- Chordae tendineae rupture (fibroelastic deficiency: FED)
- Myxomatous degeneration with prolapse and/or flail (FED +, forme fruste, Barlow's disease)
- Rheumatic heart disease
- Endocarditis
- Papillary muscle rupture (eg. post MI)
- Mitral annular calcification

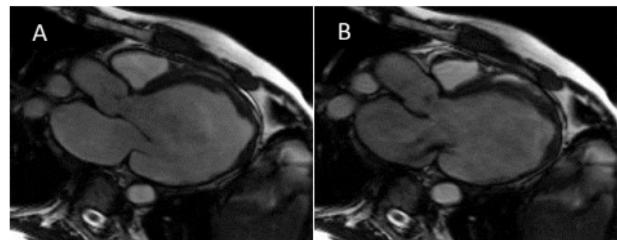
## Functional (secondary) mitral regurgitation

Occurs despite a structurally normal mitral valve

- Ischaemic cardiomyopathy
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Severe left atrial dilation



**Cine 3CH:** Prolapse of the anterior mitral leaflet (A) leading to severe mitral regurgitation in telesystole (B). Note the volume overloaded LV and the severely dilated LA



**Cine 3CH:** Severely dilated LV with an akinetic and thinned inferolateral wall (A). Severe mitral regurgitation due to tethering of both, but particular of the posterior mitral valve leaflet (B).

## Tips & Tricks

### 1. Jet direction

- **Central** (coaptation failure, symmetric tethering)
- **Eccentric** (prolapse, flail, asymmetric tethering, SAM)
  - **Anterior:** prolapse / flail posterior, tethering anterior
  - **Posterior:** prolapse / flail anterior, tethering posterior

### 2. Dilated LV suggests decompensation secondary to MR or vice versa

### 3. Dilated LA suggests chronic mitral regurgitation

### 4. Round MV annulus (diameter on 3CH and commissural 2CH view similar in size) suggests severely dilated annulus due either to LV or to LA dilatation

### 5. Tethered MV leaflets

- Asymmetric: most common posteriorly due to inferolateral MI affecting the papillary muscle
- Symmetric: severely dilated LV or secondary to rheumatic fever

6. **Regurgitant Volume of >55 mL and Regurgitant Fraction >40%** predict symptom progression and other indications for surgery

7. There is a strong correlation between post-surgical LV remodelling and MR severity as assessed by CMR

8. Consider "automatic valve tracking" (if available)

## Quantification

- **Regurgitant Volume (ml) = LV SV – SV from phase contrast VENC above AV** (not valid in the presence of a VSD)
- **Regurgitant Volume (ml) alternatively = LV SV – RV SV** (only valid in single valve disease)
- **Regurgitant Fraction (%) = RegV / LV SV x 100**

# Mitral stenosis

## Causes

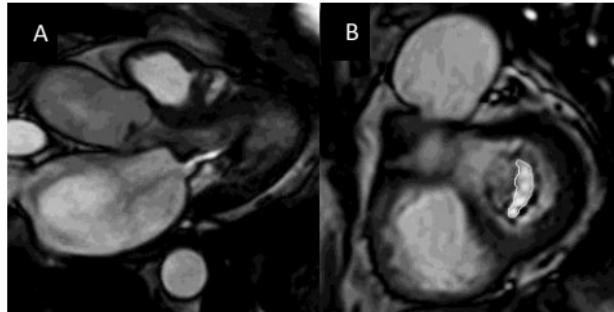
- Rheumatic valve disease
- Systemic lupus erythematosus
- Degenerative
- Congenital

## Quantification

- MV orifice area planimetry ( $\text{cm}^2$ )
- MV pressure gradients unreliable because of motion of MV apparatus

## Tips & Tricks

1. **Rheumatic MS:** fusion and thickening at the tips of the leaflets results in 'doming' of the MV leaflets ('hockey-stick').
2. **Degenerative MS:** Annular thickening and calcification affect the base and body of MV leaflet while sparing the tips
3. **Progressive LA dilatation** is marker of increased LV filling pressure and worsening MS
4. **Advanced MS** has adverse impact on RV systolic function (secondary to pulmonary hypertension)



## Rheumatic Mitral stenosis

- **Cine 3CH (A):** Restrictive opening of the thickened MV leaflets particular the posterior one. The classical sign of 'hockey stick' can be seen for the anterior MV leaflet.
- **Cine SA (B):** MVA planimetry

**Protocol**

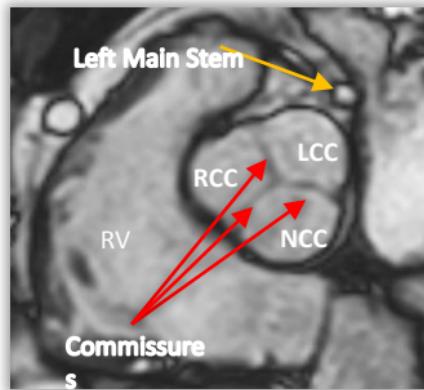
- Anatomy / LV function / RV function**
- Optimized **cine views** for AV:
  - Slice thickness 5mm - consider overlapping
  - Standard long-axis views – coronal and sagittal LVOT
  - Short axis views through the AV
- Phase contrast velocity encoded– SA** AV en-face views and sagittal oblique/coronal LVOTs

**Report**

- LV dimensions, mass (corrected for BSA) and function - global LV dysfunction is poor sign for both AR and AS
- Aortic valve morphology
- Aortic regurgitation volume (RegV) and fraction (RegF)
- Aortic stenosis
  - AV orifice area planimetry ( $\text{cm}^2$ )
  - AV peak and mean velocity through the AV
- Any associated aortic root dilatation

**Anatomy**

- AV components**
  - Three equal cusps: left coronary cusp (LCC), right coronary cusp (RCC), non-coronary cusp (NCC)
  - These cusps overlap by 2-3mm to form commissures
- Aortic Sinus**  
The 3 AV cusps attach to three adjacent aortic sinuses which have the same anatomical names: right, left, non-coronary aortic sinuses



# Aortic regurgitation

## Causes of AR

- Degenerative (most common with age)
- Dilated aortic root (e.g. hypertension, Marfan syndrome, inflammatory/aortitis)
- BAV (bicuspid aortic valve)
- Infective endocarditis or rheumatic fever

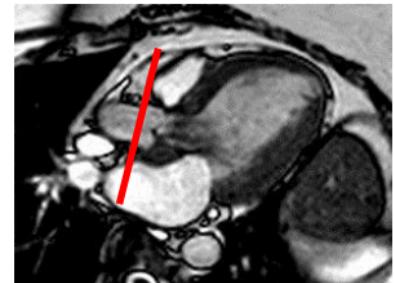
Aortic dissection

## Tips & Tricks

1. **Scan:** Position image slice close to the AV to minimize underestimation of AR (Fig: orange lines)
2. **Jet direction**
  - **Central** (aortic sinus dilatation, symmetric tethering)
  - **Eccentric** (prolapse, flail, asymmetric tethering, BAV)
4. **Aortic assessment** is likely to identify the cause of AR
5. **RegF of >33%** predict symptom progression and indications for corrective surgery\*
6. Consider flow measurement in descending aorta to detect (holo)diastolic backflow

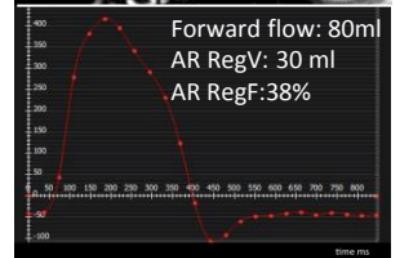
## Quantification

- **Regurgitant Volume (ml):** Through-plane phase contrast (ml)
- **Regurgitant Volume (ml) alternatively = cine LV SV – cine RV SV** (only valid in single valve disease)
- **Regurgitant Fraction (%) = RegV / Forward flow x 100**



### Cine 3CH:

Severely dilated, eccentric hypertrophied LV due to severe AR



**VENC ascending aorta:** Severe holo-diastolic backflow into the LV

**Causes of AS**

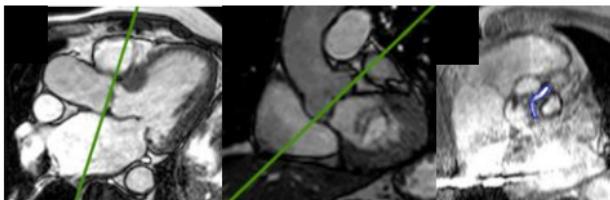
- Degenerative (most common with age)
- BAV (most common in age < 40yrs)
- Rarely rheumatic (in the developed world)

**Tips & Tricks**

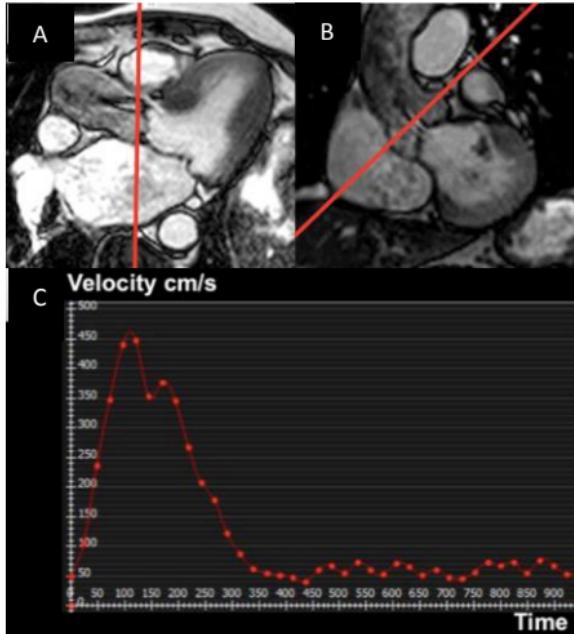
- **Scan:** Position phase contrast image slice at 90° to the stenotic jet (red lines in Fig A and B)
- Most accurate method is planimetry
- CMR can under-estimate peak velocity due to partial volume and lower temporal resolution
- Patchy mid-wall fibrosis in conjunction with LV hypertrophy is a prognostic sign in aortic stenosis

**Quantification**

- AV orifice area planimetry ( $\text{cm}^2$ )
- Peak and mean velocity assessment (m/sec)



**Aortic valve area planimetry:** cine stack parallel to the annulus



Maximal velocity assessment in a case of degenerative aortic stenosis: Projection plane in **3CH (A)** and **coronal LVOT (B)** orthogonal to the aortic stenosis jet. **Velocity time curve (C)** with a maximal velocity of 4.5 m/sec.

# Tricuspid and pulmonary valves

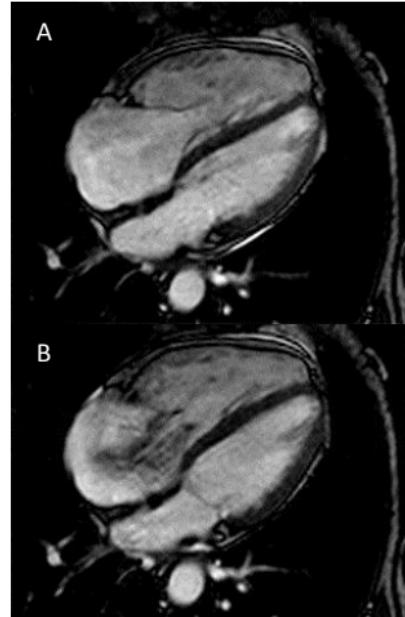
80

## Pulmonary Valve

- |                                |  |
|--------------------------------|--|
| <b>Anatomy</b>                 | <ul style="list-style-type: none"><li>• Consists of three semi-lunar cusps: anterior, left and right</li></ul>                                     |
| <b>Scan</b>                    | <ul style="list-style-type: none"><li>• RVOT views (2-orthogonal planes)</li><li>• Through-plane PV cine and phase-contrast flow</li></ul>         |
| <b>Pulmonary regurgitation</b> | <ul style="list-style-type: none"><li>• Regurgitation volume/fraction from phase contrast VENC above pulmonary valve</li><li>• RVSV–LVSV</li></ul> |

## Tricuspid Valve

- |                                |   |
|--------------------------------|---|
| <b>Anatomy</b>                 | <ul style="list-style-type: none"><li>• TV components: leaflets, annulus, sub-valvar apparatus, and RV wall contraction. Any may be involved in dysfunction of the TV.</li><li>• Leaflets: anterior, posterior and septal</li></ul> |
| <b>Scan</b>                    | <ul style="list-style-type: none"><li>• 4CH, RV 2-chamber, and RVOT views</li><li>• Through-plane TV cine can show the leaflets, and identify coaptation defects, regurgitant orifices or valve stenosis.</li></ul>                 |
| <b>Tricuspid regurgitation</b> | <ul style="list-style-type: none"><li>• SV from phase contrast VENC above pulmonary valve – LV SV</li><li>• Alternatively RV SV – LV SV</li></ul>   |



## Carcinoid heart disease

- Cine 4CH in diastole (**A**) and systole (**B**)
- The tricuspid leaflets are thickened and severely reduced in their mobility leading to free tricuspid regurgitation

**Protocol**

1. **Anatomy / LV function**
2. **Phase contrast velocity encoding**
3. **Sagittal oblique aorta SSFP cines** (candy cane view)
4. **Aortic valve cine stack**
5. **LGE** if relevant (arteritis)
6. **MR angiogram**

**Report**

1. **Dimensions: aortic root**
  - Annulus, Sinuses of Valsalva, ST junction
2. **Dimensions: Asc/Desc Ao**
  - Asc Ao at level of PA
  - Aortic arch, usually between left carotid and subclavian a.
  - Desc Ao at level of PA and diaphragm
2. **Aorta position** (left or right) and **tortuosity**
3. **Atherosclerosis, aneurysm, dissection, inflammation**
4. **Aortic flow**
5. **Associated aortic valvular stenosis or regurgitation**

**Key Points**

1. CMR is the method of choice for **non-acute aortic diseases**
2. **Standardize protocol:**
  - Measure in **end-diastole** from **cine imaging** or MRA
  - Use **same slice thickness** (<7mm) for cines
  - **Aortic root** (from 2 orthogonal LVOT cines or AV stack)
  - **Asc / desc Ao** (from sagittal oblique aorta cines or MRA)

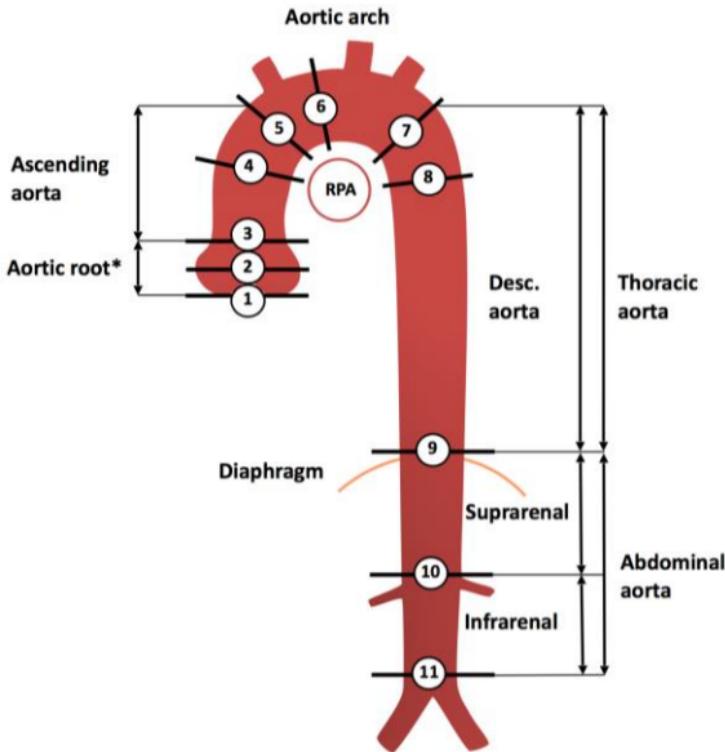
**Tips & Tricks**

Be aware of following caveats:

- LVOT / oblique views are not planed through the centre of the aorta
- MRA is usually ungated and averages pulsating aortic dimensions (i.e. not end-diastole)
- Different “windowing” of MRA
- Angled view of aorta, if taken from transaxial stack
- Inclusion of aortic wall, if taken from BB images

# Aortic diameters

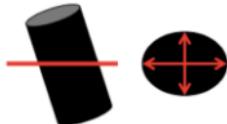
82



1. Aortic annulus
2. Sinuses of Valsalva
3. Sinotubular junction
4. Mid ascending aorta (level of the pulmonary arteries)
5. Proximal aortic arch (at the origin of the brachiocephalic trunk)
6. Mid aortic arch (btw. left common carotid and subclavian arteries)
7. Proximal descending thoracic aorta
8. Mid descending aorta (level of the pulmonary arteries)
9. At diaphragm
10. At the celiac axis origin
11. Immediately proximal to aortic bifurcation.

# Caveats of aortic measurements

## Transaxial



Overestimation due to:

- non-orthogonal plane

## Black Blood



Overestimation possible due to:

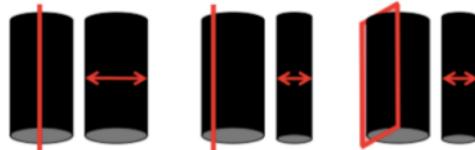
- inclusion of aortic wall

## 3D whole heart

Over- / underestimation due to:

- Lower spatial resolution
- Motion artefacts

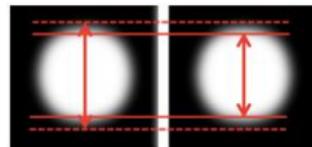
## Oblique sagittal



Underestimation due to:

- non-central or non-perpendicular plane

## MRA

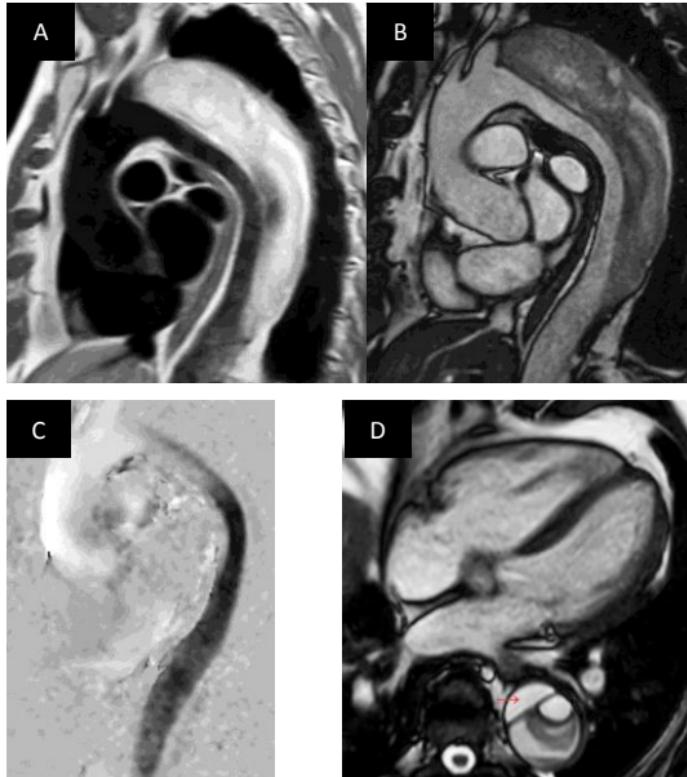


Over- / underestimation due to:

- Lower spatial resolution
- Motion artefacts, particular at aortic root / Ascending aorta
- Non-ECG triggering

# Aortic disease: Aortic dissection

84



## 60-year-old man with chronic type B dissection

- **T1w black blood candy cane view (A):** dissection originating from the aortic arch and running down the descending aorta. The false lumen is typically larger than the true lumen and is partially thrombosed
- **Cine images in candy cane view (B):** higher flow in true lumen produces higher signal than in false lumen. Inhomogeneous signal in false lumen consistent with thrombus.
- **Flow velocity encoded imaging in candy cane view (C):** flow acceleration in the true lumen
- **Cine 4CH:** the smaller true lumen (red arrow) can be easily identified by the pulsating flow. Note the organized thrombus in the false lumen.

## Protocol

1. High resolution anatomy
2. Cine imaging in all standard and targeted planes
3. Consider Tagging
4. In at least 2 optimized orthogonal planes
  - T1w black blood images +/- fat suppression
  - T2w black blood images
  - First pass myocardial perfusion imaging
  - EGE and LGE

## Report

1. Location and 3 dimensional size
2. Relation to peri-/ myocardium, valves
3. Signal intensity on T1, T1 fat sat, T2 and STIR
  - Homogenous or heterogeneous
  - Hyper-/iso-/hypointense to myocardium
4. Margins: smooth, irregular, infiltrating, pediculated
5. Specify motion with myocardium / pericardium
6. Presence and location of LGE
7. Presence of effusion (pericardial or pleural)

## Tips & Tricks

1. Consider T1 and T2 mapping
2. CMR allows tissue characterisation, but cannot provide histopathologic information.

## Key Points

1. Cardiac metastatic lesions are up to 1000 times more common than primary tumours
2. Common sources of metastatic lesions
  - Melanoma, thyroid, breast, renal, soft tissue, lung, oesophageal, hepatocellular carcinoma
3. Common benign primary tumours (70%)
  - Myxoma, lipoma, fibroelastoma, fibroma, rhabdomyoma, hemangioma
4. Common malignant primary tumors (30%)
  - Angiosarcoma, rhabdomyosarcoma, mesothelioma, fibrosarcoma, lymphoma
5. Typical tumour locations:
  - Any chamber: Lipoma (intramural / intracavitory), Haemangioma (intracavitory), Rhabdomyosarcoma (intramural, metastases)
  - Ventricles: Fibroma (intramural), Rhybdomyoma (intramural)
  - Valvular: Fibroelastoma, Vegetations.
  - Left Atrium: Thrombus, Myxoma, Fibrosarcoma, Osteosarcoma, Leiomyosarcoma (posterior wall), undifferentiated sarcoma
  - Right Atrium: Angiosarcoma, Lymphoma
  - Pericardium: Pericardial cyst, metastases

# Cardiac Masses

86

## Mass Localization

T1-w  
Transaxial  
Stack

T1-w FSE (8-10mm) free-breathing slices covering the entire thorax

or

Thinner slice (4-6mm) high-resolution acquisition in good breath-holders



Cine

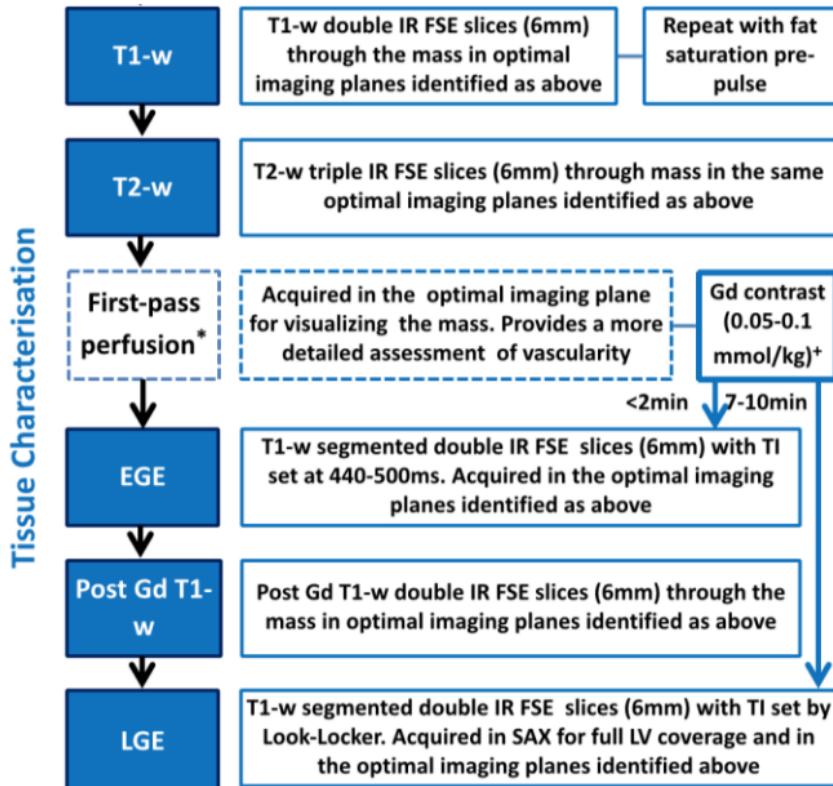
SSFP imaging in 2CH, 4CH, SAX planes.  
Additional stacks in at least 2 orthogonal customised imaging planes that optimally visualize the mass.



Tagging\*

Useful in masses that appear to infiltrate myocardium or involve pericardium

# Cardiac Masses

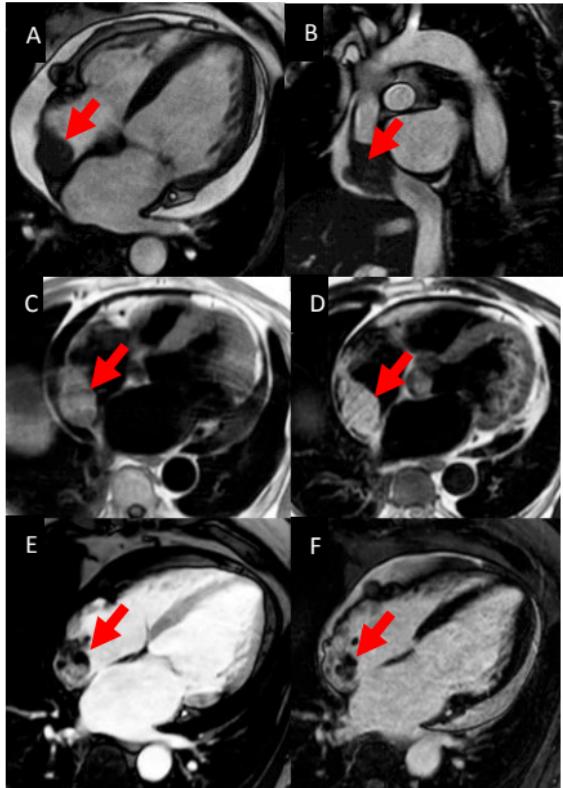


# Cardiac Masses – Tissues Characteristics

Cardiac Mass	T1w*	T2w*	LGE
<b>Pseudotumors</b>			
Thrombus	Low (high if recent)	Low (high if recent)	No uptake†
Pericardial cyst	Low	High	No uptake
<b>Benign</b>			
Myxoma	Isointense	High	Heterogeneous
Lipoma	High‡	High‡	No uptake
Fibroma	Isointense	Low	Hyperenhancement
Rhabdomyoma	Isointense	Isointense/high	No/minimal uptake
<b>Malignant</b>			
Angiosarcoma	Heterogenous	Heterogenous	Heterogeneous
Rhabdomyosarcoma	Isointense	Hyperintense	Homogeneous
Undifferentiated sarcoma	Isointense	Hyperintense	Heterogeneous/Variable
Lymphoma	Isointense	Isointense	No/minimal uptake
Metastasis §	Low	High	Heterogeneous
Melanoma (metastasis)	Hyperintense (paramagnetic metals bound by melanin)	Hypointense	

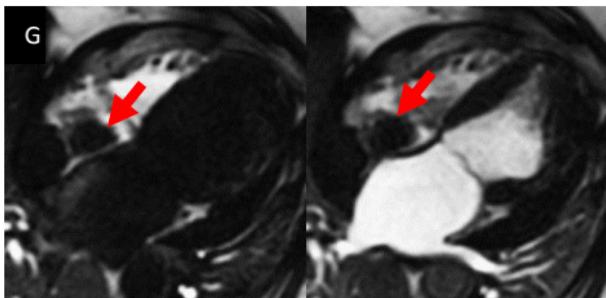
\* T1w and T2w imaging signal is given relative to myocardium; † best seen on early gadolinium enhancement imaging (no uptake) 2 minutes after contrast (Figure 1); ‡ similar to surrounding fat signal and characterized by marked suppression with fat-saturation pre-pulse. § the exception is metastatic melanoma which has a high T1w and a low T2w signal.

# Cardiac Masses – RA myxoma



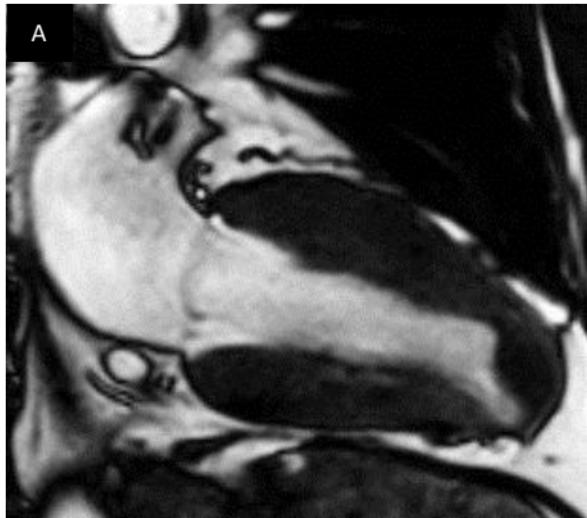
## 65-year-old woman with RA myxoma

- **Cine 4CH (A) and oblique (B) views:** mass with an irregular border attached to the posterior wall of the RA; global pericardial effusion
- **Tumour characterisation:**
  - **T1w 4CH (C):** low signal intensity compared to the myocardium
  - **T2w 4CH (D):** high signal intensity
  - **EGE 4CH (E):** heterogeneous signal intensity
  - **LGE 4CH (F):** heterogeneous signal intensity
- **First-pass perfusion (G):** heterogeneous nature of flow of contrast suggesting vascularity within the mass.



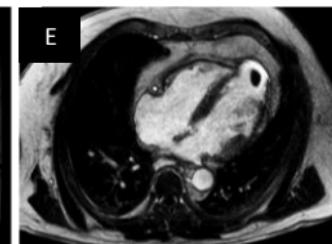
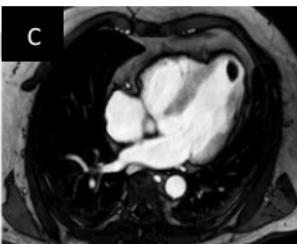
# Cardiac Masses – LV Thrombus

90

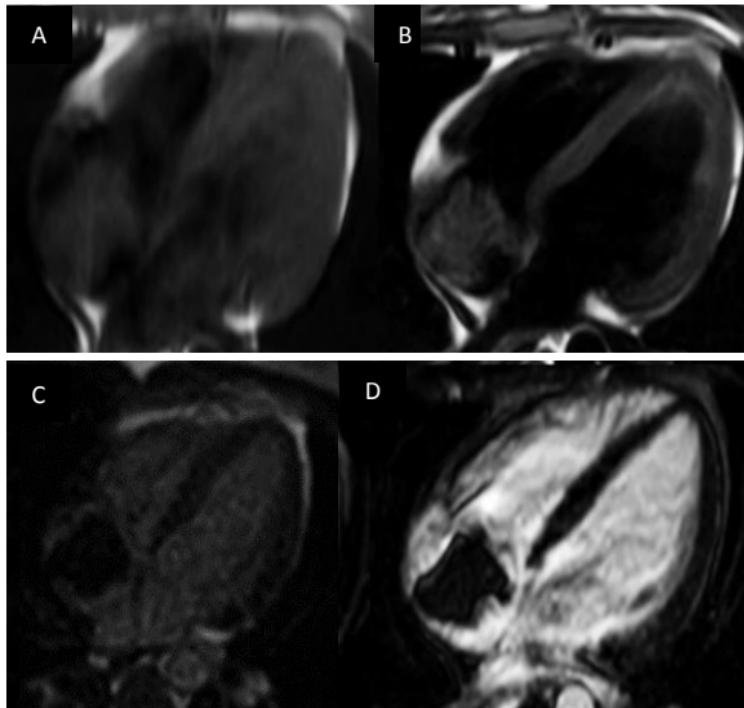


**65-year-old man with recent anterior myocardial infarction**

- **Cine 2CH (A):** apical mass situated in a dyskinetic region
- **EGE 2CH (B), 4CH (C) and LGE 2CH (D), 4CH (E):** apical mass with no uptake of contrast, typically in the area of a transmural scar.



# Cardiac Masses – Atrial Thrombus

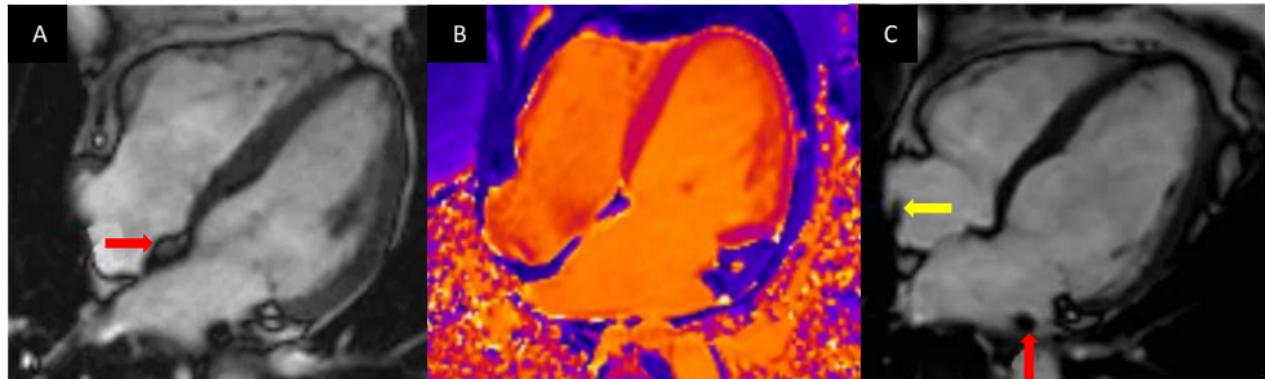


**65-year-old man with shortness of breath**

- **T1w 4CH (A):** mass in right atrium with intermediate signal intensity. The mass appears to be attached to the roof of the RA, but probably extends to the RA from the vena cava. The surface is smooth with a few mobile attachments
- **T2w 4CH (B):** the same lesion is seen showing low signal intensity
- **First pass perfusion 4CH (C):** no enhancement on first pass perfusion, suggesting its avascular nature.
- **LGE 4CH (D):** the mass shows homogeneous signal suppression
- The signal characteristics are suggestive of a **right atrial thrombus**.

# Cardiac Masses - Pseudotumours

92



**Cine 4CH (A): Lipomatous hypertrophy of the interatrial septum.** Thickening of interatrial septum (arrow); usually occurs in older and obese people; the fatty nature of such masses can be seen on fat-suppressed imaging.

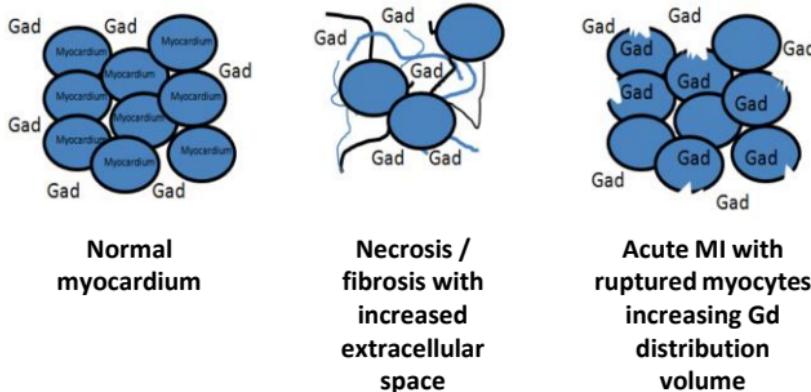
**Native T1 mapping 4CH (B): Lipomatous hypertrophy of the interatrial septum;** low native T1 values of septum ("barbell sign", sparing the fossa ovalis).

**Cine 4CH (C): Crista terminalis** (yellow arrow) divides the smooth atrial tissue from the trabeculated part of the RA.

**Coumadin ridge** (red arrow): junction of the LA appendage and the left superior pulmonary vein.

**General**

- LGE** is not disease specific and can be caused by ischaemic and non-ischaemic necrosis/fibrosis, inflammatory or infectious pathology or tumorous lesions.
- Most current gadolinium-based contrast agents are extracellular and extravascular. They do not enter intact myocytes, they accumulate within the increased extracellular space or into ruptured myocytes.

**Coronary pattern**

From subendocardium to subepicardium (ischaemic wave front) with increasing coronary occlusion time

Circumferential extent related to size of perfusion bed and occluded coronary artery

Acute reperfused MI can be associated with MVO

**Non-coronary pattern**

Subendocardium spared

Often more patchy distribution, localized in mid-wall and subepicardium

Global subendocardial LGE (uncommon even with diffuse CAD)

# LGE Patterns

## Coronary pattern

1. **Subendocardial infarct**
  - LGE transmurality correlates with likelihood of recovery after revascularization
  - LGE identifies arrhythmogenic substrate (mapping for ablation)
2. **Transmural myocardial infarct**
  - Degree of transmurality correlates inversely with viability
3. **Ischaemic cardiomyopathy**
  - Often also LV dilatation, differential diagnosis of DCM
  - Coronary LGE pattern with subendocardial/transmural LGE
  - LGE identifies arrhythmogenic substrate (mapping for ablation)



Subendocardial      Transmural

## Non-coronary pattern

1. **Cardiac amyloidosis**
  - Difficulty in nulling myocardial signal
  - Global subendocardial distribution (non-coronary pattern) or patchy subendocardial or transmural LGE
  - Base-apex LGE gradient, blood pool early darkening
  - **DD AL vs. ATTR amyloidosis**
    - AL: less extensive LGE, often (global) subendocardial pattern, QALE score < 13
    - ATTR: more extensive and diffuse LGE, often more diffuse and transmural pattern, QALE score ≥ 13



Global subendocardial      Patchy subendocardial

**2. Dilated cardiomyopathy**

- Diffuse mid-wall or subepicardial septal LGE (reflecting fibrosis and not matching a coronary perfusion bed)
  - Often localized to the inferoseptal wall
  - In most cases of DCM there is no LGE -> consider T1 mapping
- 

**3. Hypertrophic cardiomyopathy**

- Focal (mid-wall) LGE of anterior and posterior RV insertion points (hinge points) and of hypertrophied segments
  - Apical HCM: apical wall may develop ischaemia with wall thinning → LGE can be transmural (mimicking ischaemic injury in the absence of epicardial CAD).
  - LGE in HCM indicates replacement fibrosis but may also indicate necrosis/scarring
- 

**4. Systemic Sclerosis**

- Basal to mid-interventricular septum and RV insertion points or diffuse patchy LGE
- 

**5. Myocarditis**

- Lateral, typically inferolateral or inferior wall with a mid-wall to subepicardial enhancement
- Atypical cases of myocarditis with transmural or diffuse LGE
- Often concomitant LGE involvement of the pericardium



# LGE Patterns

## 6. Anderson-Fabry Disease

- Mid-wall (or subepicardial) LGE of the mid to basal inferolateral wall
- Consider T1-Mapping: low ECV and low native T1 value of septum



## 7. Sarcoidosis

- Basal and mid-interventricular septum but also patchy LGE



## 8. ARVC

- Diffuse LGE of RV (and occasionally LV) wall



## 9. Endomyocardial fibrosis (Loeffler's endocarditis)

- Restrictive CMP with endocardial fibrotic tissue deposition of inflow tract and apex of one or both ventricles
- "V sign" of LGE pattern at ventricular apex, characterized by a 3-layer appearance of dark myocardium, bright thickened fibrotic endocardium, and overlying dark thrombus

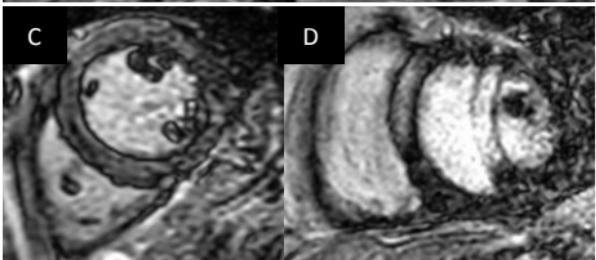
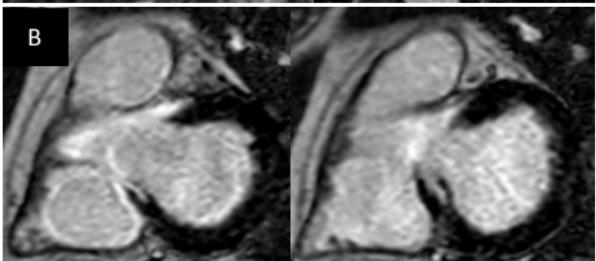
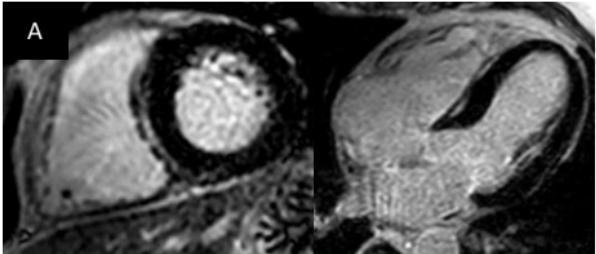


## 10. False positive LGE

- Septal perforator (mimicking septal basal mid-wall enhancement)
- RV trabeculation (contrast extending underneath the muscle mimicking septal enhancement)



# Pitfalls in reporting septal LGE



**Not all septal hyperenhancement is myocardial fibrosis!**

- Septal pseudo-enhancement due to RV blood pool interposed between RV trabeculations and interventricular septum (**A**). A similar pseudo-enhancement may appear due to large septal perforator arteries.
- Normal hyperenhancement of the fibrous skeleton of the heart in the LVOT region (**B**)
- Unsuccessful nulling of the myocardium caused by choosing an incorrect inversion time (**C**)
- Ghosting artefact due to poor breath-holding (**D**)

## Tips & Tricks

1. Modify the phase encoding direction. Real myocardial fibrosis should be present irrespective of the phase encoding direction.
2. Acquire images in orthogonal planes through the suspected hyperenhancement areas. Real fibrosis should be seen in both original and perpendicular planes.
3. In suspected septal perforator arteries, follow its trajectory to the LAD in the septum.
4. Compare LGE images with cines to identify RV trabeculation and blood pool.

# LV Recesses / Outpouchings

98

## LV Crypt (congenital)

### 1. Definition\*

- Narrow V/U shaped **fissure(s) confined to compacted myocardium** and penetrating >50% of wall, perpendicular to LV long axis

### 2. Contractility

- Systolic obliteration

### 3. Segment

- Inferior / inferoseptal basal / mid wall

### 4. LGE

- No LGE

### 5. Clinical relevance

- Isolated clefts in normal hearts
- Early biomarker in HCM mutation carriers without LV hypertrophy
- The terms cleft, crypt, crevices and fissures are used interchangeably.



## LV Non-Compaction (congenital)

### 1. Definition

- Hypertrabeculated, non-compacted myocardium
- Deep intertrabecular recesses

### 2. Contractility

- LV dilatation and dysfunction

### 3. Segment

- Apex, lateral wall, inferior wall

### 4. LGE

- LGE possible

### 5. Clinical relevance

- Very difficult diagnosis; may not be based on imaging criteria alone
- Poor prognosis
- Heart failure
- Arrhythmias (SCD)
- Thromboembolism



# LV Recesses / Outpouchings

## True aneurysm (infarction, congenital)

### 1. Definition

- Wide-necked, thin (scarred) outpouching of all layers of the myocardium beyond epicardial contour

### 2. Contractility

- Dyskinesia (systolic out-bulging) or akinesia

### 3. Segment

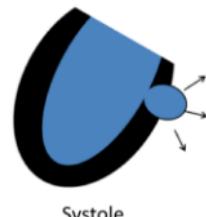
- Mainly LAD territory, particularly LV apex

### 4. LGE

- Transmural LGE in a coronary pattern

### 5. Clinical relevance

- Thrombus formation
- Heart failure
- Arrhythmias
- May be difficult to differentiate from pseudoaneurysm



## Pseudoaneurysm (infarction, trauma)

### 1. Definition

- Narrow-necked myocardial rupture contained by the pericardium

### 2. Contractility

- Dyskinesia (systolic out-bulging) or akinesia

### 3. Segment

- Mainly inferior (RCA) and lateral (CX) wall

### 4. LGE

- Perianeurysmal LGE in a coronary pattern but no LGE in the pericardial sac

### 5. Clinical relevance

- 30-45% risk of rupture with high mortality rate
- May be difficult to differentiate from true aneurysm



# LV Hypertrophy – differential diagnosis

100

## General

- Echocardiography remains the first line imaging modality to assess LVH.
- In most cases, LVH may be attributable to hypertension, aortic stenosis, or obesity.
- Not considered in this chapter: Mitochondrial myopathy and mucopolysaccharidosis.

## Athlete's Heart versus HCM

Data feature	Athlete's Heart	HCM
Increased wall thickness	Yes	Yes
Atrial remodeling	Yes	Yes
Systolic function	Normal	Normal
Mechanical function (strain)	Normal	Abnormal
Diastolic physiology	Normal	Abnormal
ECV	Low	High

## Athlete's Heart

### 1. History

- High level sporting activity

### 2. ECG

- Resting bradycardia
- I° AV block
- High voltage QRS
- Early repolarisation

### 3. Anatomy/Function

- Increased chamber dimensions and LV wall thickness
- Symmetric LVH
  - Endurance athletes: eccentric LVH
  - Strength-trained athletes: concentric LVH
  - Combination athletes: overlapping features of endurance and strength-trained athletes
- Max. wall thickness  $\leq 14$  mm
- Black athletes show greater increases in LV wall thickness
- Normal systolic function
- Normal mechanical function (strain)
- Normal diastolic function

### 4. LGE pattern

- No LGE or non-specific pattern

### 5. Follow up

- LVH $\downarrow$  after deconditioning

# LV Hypertrophy – differential diagnosis

## Aortic stenosis

### 1. History

- Severe aortic stenosis

### 2. ECG

- High voltage QRS
- LV strain pattern

### 3. Anatomy/Function

- **Symmetric concentric LVH:**
  - Degree of hypertrophy associated with the degree of stenosis
  - LVOT obstruction uncommon
- **Normal systolic function**
  - LV dilation and heart failure in decompensated state
  - Abnormal mechanical function (strain)
  - Diastolic dysfunction

### 4. LGE pattern

- Non-specific LGE pattern: patchy, diffuse or septal mid-wall LGE

### 5. T1 mapping

- Native T1↑
- ECV↑

### 6. Follow up

- LVH↓ and fibrosis↓ after aortic valve replacement

## Hypertensive heart disease

### 1. History

- Long-lasting hypertension

### 2. ECG

- High voltage QRS
- LV strain pattern

### 3. Anatomy/Function

- **Symmetric concentric LVH:**
  - Max. wall thickness <15 mm (Caucasian), <15-20 mm (Black)
- **Basal septum hypertrophy**
  - Common in elderly and hypertensive patients
- LVOT obstruction uncommon
  - Normal systolic function – in late stages systolic dysfunction can develop
- Abnormal mechanical function (strain)
- Diastolic dysfunction

### 4. LGE pattern

- Occasionally patchy LGE pattern or focal (mid wall) LGE of RV insertion points (hinge points)

### 5. Follow up

- LVH↓ under OMT for hypertension

## General

- CMR is the reference standard for calculation of RV volumes, mass, flow and function.
- Multiparametric properties of CMR allow for tissue characterization and aid in the differential diagnosis of dilated RV pathologies.

*Not considered in this chapter: congenital diseases other than ASD and partial anomalous pulmonary venous drainage. For more information: see Congenital Heart Disease Pocket guide.*

## The right ventricle

- Complex geometrical shape
- More (coarse) trabeculation and thinner walls (3-5 mm), less compacted myocardium than LV
- RV ejection is mostly determined by longitudinal rather than circumferential contraction

## Protocol

- Anatomy**
- LV function** (including atria): LV cine stack, 4/2/3 CH cines
- RV function:** transaxial RV cine stack, RVOT, RV in-/outflow and RV 2CH cines
- MPA, PAs cines**
- MPA and Ao flow**
- Whole Heart or MRA (to delineate pulmonary veins and to exclude PV stenosis post ablation)
- LGE**

## Ventricular interdependence

- Mediated by septum
- RV is more sensitive than LV to volume and pressure loading
- RV volume overload → diastolic D-shaping\*
- RV pressure overload → systolic D-shaping\*

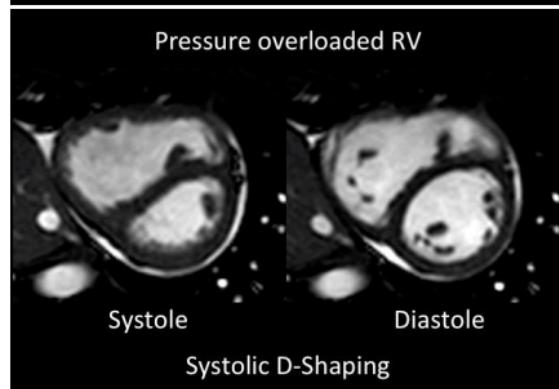
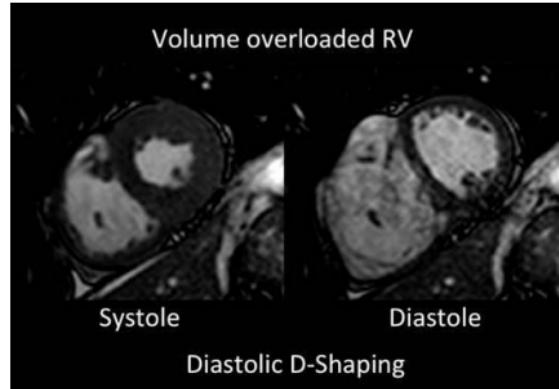
\*Overlapping features possible

**RV Infarction**

- Up to 50% of inferior MI (proximal to acute marginal RCA branches) also involve the RV; isolated RV infarction is rare
- RV infarction not limited to RCA but also occurs in LAD and CX infarction and rarely in the absence of coronary disease in substantial RV hypertrophy
- Findings: RV RWMA, impaired RV function
- Myocardial oedema on T2w imaging in acute RV infarction
- Check for RV thrombus with subsequent PE
- LGE: Enhancement in RV +/- RCA territory of LV

**Non-ischaemic cardiomyopathy**

- Various non-ischaemic cardiomyopathies may cause RV dilatation, please see specific chapters



## General

- RV diastolic volumes ↑
- RV function may be impaired
- Normal RV free wall thickness
- Diastolic D-shaping / bounce of the septum due to rapid filling of the RV in diastole (diastolic pressure ↑)
- Fibrosis at RV insertion points possible

## Severe pulmonary regurgitation

- Rare, but frequently after post-Fallot repair or pulmonary valvotomy
- Dilated RV / RVOT / MPA / PAs (Ao > MPA diameter in healthy subjects)
- Severe PR jet is laminar and difficult to visualise
- Valve assessment – see specific chapter

## Severe left / right shunt (ASD and/or PAPVD)

- Check for Ebstein's anomaly (frequently associated)
- Dilated RA, RV, MPA, and PAs (usually Ao > MPA)
- Dilated inferior vena cava / coronary sinus
- Percutaneous closure – check for sufficient rim surrounding most of the defect to lodge the device, particular inferiorly
- **Red flags:**
  - Significant RV dilatation / dysfunction
  - Qp : Qs > 1.8
  - Pulmonary hypertension

## Severe tricuspid regurgitation

- Often secondary due to annulus dilation (RV and/or RA dilatation)
- Always check for Ebstein's anomaly
- Severe TR jet is laminar and difficult to visualise
- Dilated inferior vena cava / coronary sinus
- Valve assessment – see specific chapter

## General

- RV dilation and dysfunction in progressive disease
- Hypertrophy of the RV free wall ( $>5$  mm) and of the interventricular septum
- Systolic D-shaping if severe pressure overload
- Fibrosis at RV and RV insertion points

## Severe pulmonary hypertension

- Dilated MPA, and PAs (Ao  $>$  MPA diameter in healthy subjects)
- Classification
  - PH due to left heart disease (systolic and/or diastolic LV dysfunction, valvular disease, etc)
  - Chronic thromboembolic PH
  - Lung disease and/or hypoxia
  - Pulmonary arterial hypertension
  - Other causes

## Severe pulmonary stenosis

- Valvular, sub-valvular, supra-valvular
- Congenital stenosis (mainly Fallot's tetralogy)
  - → Typically mobile leaflets with fused tips (Prussian helmet sign)
- Rarely: rheumatic and carcinoid
  - → Thickened valve with restricted movement
- Often post-stenotic dilatation of MPA and PAs with preferential dilatation of the LPA (vs. RPA): LPA with in-line orientation; RPA takes a more right angle course from the MPA
- Valve assessment – see specific chapter

# LV Parameters Male

106

	20-29 (years)	30-39 (years)	40-49 (years)
<b>EDV (ml)</b>	167 (126, 208)	163 (121, 204)	159 (117, 200)
<b>ESV (ml)</b>	58 (35, 80)	56 (33, 78)	54 (31, 76)
<b>SV (ml)</b>	109 (81, 137)	107 (79, 135)	105 (77, 133)
<b>EF (%)</b>	65 (57, 74)	66 (57, 75)	66 (58, 75)
<b>Mass (g)</b>	148 (109, 186)	147 (109, 185)	146 (108, 185)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	86 (68, 103)	83 (66, 101)	81 (64, 99)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	30 (19, 41)	29 (18, 39)	27 (17, 38)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	56 (44, 68)	55 (43, 67)	54 (42, 66)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	76 (59, 93)	75 (59, 92)	75 (58, 91)

	50-59 (years)	60-69 (years)	70-79 (years)
<b>EDV (ml)</b>	154 (113, 196)	150 (109, 191)	146 (105, 187)
<b>ESV (ml)</b>	51 (29, 74)	49 (27, 72)	47 (25, 70)
<b>SV (ml)</b>	103 (75, 131)	101 (73, 129)	99 (71, 127)
<b>EF (%)</b>	67 (58, 76)	67 (58, 76)	68 (59, 77)
<b>Mass (g)</b>	146 (107, 184)	145 (107, 183)	144 (106, 183)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	79 (62, 97)	77 (60, 95)	75 (58, 93)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	26 (15, 37)	25 (14, 36)	24 (13, 35)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	53 (41, 65)	52 (40, 64)	51 (39, 63)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	74 (57, 91)	73 (57, 90)	73 (56, 89)

Data presented as mean (95% confidence interval). Analysed with CMR tools software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.

# LV Parameters Female

	20-29 (years)	30-39 (years)	40-49 (years)
<b>EDV (ml)</b>	139 (99, 179)	135 (94, 175)	130 (90, 171)
<b>ESV (ml)</b>	48 (29, 66)	45 (27, 64)	43 (25, 62)
<b>SV (ml)</b>	91 (63, 119)	89 (61, 117)	87 (59, 115)
<b>EF (%)</b>	66 (56, 75)	66 (57, 75)	67 (58, 76)
<b>Mass (g)</b>	110 (74, 146)	106 (70, 142)	107 (71, 143)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	82 (65, 99)	79 (62, 96)	76 (59, 93)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	28 (19, 37)	27 (17, 36)	25 (16, 34)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	54 (42, 66)	53 (40, 65)	51 (39, 63)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	62 (47, 77)	62 (47, 77)	63 (48, 77)

	50-59 (years)	60-69 (years)	70-79 (years)
<b>EDV (ml)</b>	126 (86, 166)	122 (82, 162)	118 (77, 158)
<b>ESV (ml)</b>	41 (22, 59)	39 (20, 57)	36 (18, 55)
<b>SV (ml)</b>	85 (57, 113)	83 (56, 111)	81 (54, 109)
<b>EF (%)</b>	68 (59, 77)	69 (60, 78)	69 (60, 78)
<b>Mass (g)</b>	108 (72, 144)	109 (73, 145)	110 (74, 146)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	73 (56, 90)	70 (53, 87)	67 (50, 84)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	24 (14, 33)	22 (13, 31)	21 (12, 30)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	50 (37, 62)	48 (36, 60)	47 (34, 59)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	63 (48, 78)	63 (48, 78)	63 (49, 78)

Data presented as mean (95% confidence interval). Analysed with CMRtools software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring

# RV Parameters Male

108

	20-29 (years)	30-39 (years)	40-49 (years)
<b>EDV (ml)</b>	177 (127, 227)	171 (121, 221)	166 (116, 216)
<b>ESV (ml)</b>	68 (38, 98)	64 (34, 94)	59 (29, 89)
<b>SV (ml)</b>	108 (74, 143)	108 (74, 142)	107 (73, 141)
<b>EF (%)</b>	61 (48, 74)	63 (50, 76)	65 (52, 77)
<b>Mass (g)</b>	70 (42, 99)	69 (40, 97)	67 (39, 95)
<i>Indexed to BSA</i>			
<b>EDV/BSA (ml/m<sup>2</sup>)</b>	91 (68, 114)	88 (65, 111)	85 (62, 108)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	35 (21, 50)	33 (18, 47)	30 (16, 45)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	56 (40, 72)	55 (39, 71)	55 (39, 71)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	36 (23, 50)	35 (22, 49)	34 (21, 48)
	50-59 (years)	60-69 (years)	70-79 (years)
<b>EDV (ml)</b>	160 (111, 210)	155 (105, 205)	150 (100, 200)
<b>ESV (ml)</b>	55 (25, 85)	50 (20, 80)	46 (16, 76)
<b>SV (ml)</b>	106 (72, 140)	105 (71, 139)	104 (70, 138)
<b>EF (%)</b>	66 (53, 79)	68 (55, 81)	70 (57, 83)
<b>Mass (g)</b>	65 (37, 94)	63 (35, 92)	62 (33, 90)
<i>Indexed to BSA</i>			
<b>EDV/BSA (ml/m<sup>2</sup>)</b>	82 (59, 105)	79 (56, 101)	75 (52, 98)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	28 (13, 42)	25 (11, 40)	23 (8, 37)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	54 (38, 70)	53 (37, 69)	52 (36, 69)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	33 (20, 46)	32 (19, 45)	31 (18, 44)

Data presented as mean (95% confidence interval). Analysed with CMRtools software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring

# RV Parameters Female

	20-29 (years)	30-39 (years)	40-49 (years)
<b>EDV (ml)</b>	142 (100, 184)	136 (94, 178)	130 (87, 172)
<b>ESV (ml)</b>	55 (29, 82)	51 (25, 77)	46 (20, 72)
<b>SV (ml)</b>	87 (61, 112)	85 (59, 111)	84 (58, 109)
<b>EF (%)</b>	61 (49, 73)	63 (51, 75)	65 (53, 77)
<b>Mass (g)</b>	54 (33, 74)	51 (31, 72)	49 (28, 70)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	84 (65, 102)	80 (61, 98)	76 (57, 94)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	32 (20, 45)	30 (17, 43)	27 (14, 40)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	51 (39, 63)	50 (38, 62)	49 (37, 61)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	32 (22, 42)	30 (20, 40)	29 (19, 39)

	50-59 (years)	60-69 (years)	70-79 (years)
<b>EDV (ml)</b>	124 (81, 166)	117 (75, 160)	111 (69, 153)
<b>ESV (ml)</b>	42 (15, 68)	37 (11, 63)	32 (6, 58)
<b>SV (ml)</b>	82 (56, 108)	80 (55, 106)	79 (53, 105)
<b>EF (%)</b>	67 (55, 79)	69 (57, 81)	71 (59, 83)
<b>Mass (g)</b>	47 (26, 68)	45 (24, 66)	43 (22, 63)

*Indexed to BSA*

<b>EDV/BSA (ml/m<sup>2</sup>)</b>	72 (53, 90)	68 (49, 86)	64 (45, 82)
<b>ESV/BSA (ml/m<sup>2</sup>)</b>	24 (11, 37)	21 (8, 34)	19 (6, 32)
<b>SV/BSA (ml/m<sup>2</sup>)</b>	48 (36, 60)	46 (34, 58)	45 (33, 57)
<b>Mass/BSA (g/m<sup>2</sup>)</b>	27 (17, 37)	26 (16, 36)	24 (14, 35)

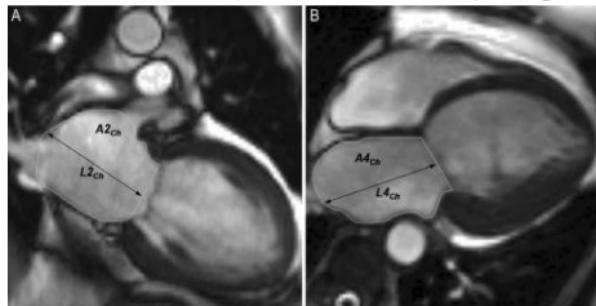
# Atrial Parameters

110

## Left atrial volumes

	Men	Women
4CH LA area (cm <sup>2</sup> )	15 – 29	13 – 27
2CH LA area (cm <sup>2</sup> )	12 – 30	10 – 28
4CH LA area/BSA (cm <sup>2</sup> /m <sup>2</sup> )	7 – 15	8 – 16
2CH LA area/BSA (cm <sup>2</sup> /m <sup>2</sup> )	6 – 16	6 – 16
LA Volume (Bi-plane) (ml)	43 – 163	47 – 131
LA Volume/BSA (ml/m <sup>2</sup> )	26 – 52	27 – 53

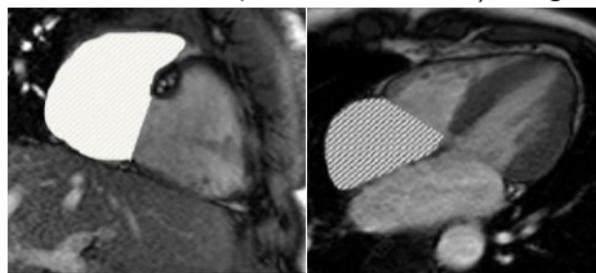
$$\text{LA Volume} = 0.85 \times (\text{Area 4CH} \times \text{Area 2CH}) / \text{Length}$$



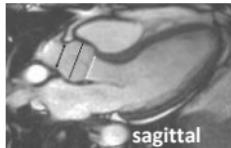
## Right atrial volumes

	Men/Women
4CH RA area (cm <sup>2</sup> )	14 – 30
2CH RA area (cm <sup>2</sup> )	14 – 30
4CH RA area/BSA (cm <sup>2</sup> /m <sup>2</sup> )	8 – 16
2CH RA area/BSA (cm <sup>2</sup> /m <sup>2</sup> )	7 – 17
RA Volume (Bi-plane) (ml)	37 – 169
RA Volume/BSA (ml/m <sup>2</sup> )	18 – 90

$$\text{RA Volume} = 0.85 \times (\text{Area 4CH} \times \text{Area 2CH}) / \text{Length}$$



# Aortic Root Dimensions



**Male**                    **20-29 (years)**    **30-39 (years)**    **40-49 (years)**    **50-59 (years)**    **60-69 (years)**    **70-79 (years)**

Annulus (s)	21.4 ± 2.4	20.7 ± 1.7	21.6 ± 2.0	22.8 ± 2.8	23.5 ± 1.8	23.3 ± 2.7
Annulus (c)	26.5 ± 1.8	25.2 ± 2.4	25.8 ± 1.5	26.4 ± 3.7	26.5 ± 1.8	26.6 ± 1.9
Aortic sinus (s)	30.5 ± 3.9	29.8 ± 3.8	32.0 ± 2.4	33.3 ± 6.1	33.6 ± 2.7	35.1 ± 3.7
Aortic sinus (c)	32.5 ± 3.4	31.8 ± 4.8	33.6 ± 2.6	34.7 ± 6.4	35.7 ± 3.3	36.1 ± 3.5
Sinotubular junction (s)	23.3 ± 3.4	22.2 ± 4.0	24.4 ± 3.3	26.6 ± 3.1	27.6 ± 3.6	28.3 ± 2.7
Sinotubular junction (c)	23.7 ± 3.5	22.2 ± 3.0	24.5 ± 2.4	26.5 ± 3.7	27.5 ± 2.4	27.8 ± 1.7

**Female**                    **20-29 (years)**    **30-39 (years)**    **40-49 (years)**    **50-59 (years)**    **60-69 (years)**    **70-79 (years)**

Annulus (s)	19.5 ± 2.4	19.2 ± 2.3	19.9 ± 2.2	20.1 ± 1.9	20.4 ± 1.1	20.2 ± 1.5
Annulus (c)	23.6 ± 3.0	22.9 ± 2.3	23.3 ± 1.5	22.7 ± 2.1	22.3 ± 1.5	23.3 ± 1.5
Aortic sinus (s)	26.5 ± 4.0	26.9 ± 3.1	31.5 ± 2.8	29.1 ± 2.5	30.1 ± 2.5	30.2 ± 2.0
Aortic sinus (c)	28.5 ± 4.9	28.2 ± 3.1	32.0 ± 2.5	30.2 ± 2.3	31.0 ± 2.7	31.3 ± 1.8
Sinotubular junction (s)	21.1 ± 3.3	21.8 ± 2.8	25.7 ± 2.3	24.1 ± 1.9	25.1 ± 3.0	25.0 ± 2.0
Sinotubular junction (c)	21.5 ± 2.7	22.1 ± 2.7	25.5 ± 2.1	23.4 ± 2.1	24.7 ± 1.6	25.1 ± 1.3

Data measured in diastole and presented as mean ± SD in mm.

Analyzed from sagittal (s) and coronal (c) SSFP LVOT cines

# Safety - Gadolinium-containing contrast agents

112

## General

- **Linear agents:** gadobenic acid (MultiHance), gadodiamide (Omniscan), gadopentetic acid (Magnevist) and gadoversetamide (OptiMARK) have the potential to release gadolinium *in vivo*, which can be retained in body tissues.
- **Macrocyclic agents:** gadoteric acid (Dotarem), gadobutrol (Gadovist), gadoteridol (Prohance) are more stable and have a much lower propensity to release gadolinium than linear agents.
- Because of concerns over tissue retention, the EMA has recommended the suspension of the marketing authorisations for linear contrast agents ([www.ema.europa.eu](http://www.ema.europa.eu)).

## Brain retention

- **Linear gadolinium based contrast agents** have been associated with gadolinium retention in the dentate nucleus and globus pallidus in the brain. To date, no signs of harm and no symptoms or diseases have been linked to this retention.

## Nephrogenic systemic fibrosis (NSF)

- **Linear gadolinium based contrast agents** have been associated with NSF in patients with severe renal impairment.
- NSF is an extremely rare but serious and potentially life-threatening condition.
- NSF is characterised by the deposition of collagen in the skin and possible organ involvement of other organs, including the lungs, liver, muscles and heart.

No cases of NSF have been reported in patients with normal renal function and macrocyclic contrast agents appear to be much less likely to cause NSF.

## Tips & Tricks

- **eGFR 30-60ml/min/1.73m<sup>2</sup>:** choose safest contrast agent, use only with caution
- **eGFR <30ml/min/1.73m<sup>2</sup>:** linear contrast agents contraindicated
- **In severe renal failure:** consider haemodialysis within 2 hours after contrast agent administration (although not proven to prevent NSF)
- See also: [www.rcr.ac.uk](http://www.rcr.ac.uk)

# Safety Common Devices

## MR unsafe

- Any device which is known to threaten or pose hazard in all MR environments
- Most pacemakers
- Insulin pumps
- Most implantable cardioverter / defibrillators
- Metal foreign bodies in the eye



## MR safe

- Any device which is known to pose **NO** hazard in **all** MR environments\*
- Only assume that a device is MR safe if it has this logo on it



## MR conditional

- Any device which is demonstrated to pose **NO** hazard in a **specific** MR environment with **specified** conditions
- Most metallic heart valves
- Intra-coronary stents
- Prosthetic joints
- Dentures



## Tips & Tricks

- If in doubt check:

**[www.mrisafety.com](http://www.mrisafety.com)**

# Common MR Terminology

114

Sequence Type	GE	Philips	Siemens
Fast Spin Echo	FSE (Fast SE)	TSE (Turbo SE)	Turbo SE
Gradient recalled echo	GRE	FFE	GRE
Spoiled gradient echo	SPGR / MPSPGR	T1 FFE	FLASH
Balanced gradient echo	FIESTA	bFFE / bTFE	TrueFISP
Gradient echo – echo planar	GRE EPI	FFE-EPI / TFE-EPI	EPIFI
Contrast enhanced MRA		Bolus Trak	Care Bolus
k-space lines	Views per segment	Turbofactor	No of segments
Parallel imaging: Image-based reconstruction	ASSET	SENSE	mSENSE
Parallel imaging: k-space-based reconstruction	ARC		GRAPPA

<b>2CH</b>	2-chamber view	<b>FOV</b>	Field of view	<b>ROI</b>	Region of interest
<b>3CH</b>	3-chamber view	<b>Gd</b>	gadolinium	<b>R-L</b>	Right - left
<b>4CH</b>	4-chamber view	<b>GFR</b>	Glomerular filtration rate	<b>RV</b>	Right ventricle
<b>AF</b>	Atrial fibrillation	<b>HCM</b>	Hypertrophic cardiomyopathy	<b>SA</b>	Short axis
<b>Ao</b>	Aorta	<b>HLA</b>	Horizontal long axis	<b>SAECG</b>	Signal averaged ECG
<b>ARVC</b>	Arrhythmogenic RV cardiomyopathy	<b>HR</b>	Heart rate	<b>SAM</b>	Systolic anterior motion
<b>Asc</b>	Ascending	<b>LAD</b>	Left anterior descending artery	<b>SD</b>	Standard deviation
<b>AV</b>	Aortic valve	<b>LGE</b>	Late gadolinium enhancement	<b>SI</b>	Signal intensity
<b>BMD</b>	Becker Muscular Dystrophy	<b>LV</b>	Left ventricle	<b>SNR</b>	Signal-to-noise ratio
<b>BSA</b>	Body surface area	<b>LVNC</b>	LV non compaction cardiomyopathy	<b>SSFP</b>	Steady-state free precession
<b>b-SSFP</b>	Balanced steady-state free precession	<b>LVOT</b>	Left ventricular outflow tract	<b>STIR</b>	Short TI inversion recovery
<b>CMR</b>	Cardiovascular Magnetic Resonance	<b>NC/C</b>	Non-compacted / compacted	<b>SV</b>	Stroke volume
<b>CX</b>	Circumflex artery	<b>NSVT</b>	Nonsustained ventricular tachycardia	<b>T1w</b>	T1-weighted
<b>DCM</b>	Dilated cardiomyopathy	<b>MR</b>	Mitral regurgitation	<b>T2w</b>	T2-weighted
<b>Desc</b>	Descending	<b>MRA</b>	Magnetic resonance angiography	<b>T2*</b>	T2 star
<b>DMD</b>	Duchenne Muscular Dystrophy	<b>MVO</b>	Microvascular obstruction	<b>TI</b>	Time from inversion
<b>EDV</b>	End-diastolic volume	<b>PA</b>	Pulmonary artery	<b>VENC</b>	Velocity Encoding
<b>EGE</b>	Early gadolinium enhancement	<b>fQRS</b>	Filtered QRS	<b>VES</b>	Ventricular extra systole
<b>EF</b>	Ejection fraction	<b>RA</b>	Right atrium	<b>VLA</b>	Vertical long axis
<b>F-H</b>	Foot- head	<b>RCA</b>	Right coronary artery	<b>WMSI</b>	Wall motion score index

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## NOTES

120





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