The clinical indications for Cardiovascular Magnetic Resonance (CMR) continue to expand. This pocket guide aims to provide a day-to-day companion for those new to CMR and for those looking for a quick reference guide in routine practice. The booklet gives an overview of established normal ranges for CMR measurements, common acquisition methods and clinical indications for CMR. For each indication we provide typical scan protocols, tips and tricks and a guide for reporting.

Bernhard Herzog  
John Greenwood  
Sven Plein

The Cardiovascular Magnetic Resonance Pocket Guide represents the views of the ESC Working Group on Cardiovascular Magnetic Resonance and 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.

For more detailed information on CMR protocols, current evidence, and with extensive examples on CMR cases we recommend the CMR-Update book, available through www.herz-mri.ch.

We acknowledge the support and advice we have received from Regina Herzog, Gavin Bainbridge, Ananth Kidambi, Manish Motwani and Akhlaque Uddin.
### 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 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

### 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

### Tips & Tricks

Any doubt? Check online: [www.mrisafety.com](http://www.mrisafety.com)
General

• Thought to be related to toxic effects of Gd ions in patients with advanced renal failure / haemodialysis
• Causes fibrosis of skin, joints, eyes, and internal organs
• Very rare, but serious syndrome

Contrast media and safety

Safest (cyclical structure):
• Dotarem, Gadovist, ProHance

Intermediate safety (ionic linear structure):
• Magnevist, MultiHance, Primovist, Vasovist

Lowest safety (linear non-ionic structure):
• Omniscan, OptiMARK

Note: No cases of NSF have been reported in patients with normal renal function

Tips & Tricks

• eGFR 30-60ml/min/1.73m²: choose safest contrast agent, use only with caution
• eGFR <30ml/min/1.73m²: linear structured contrast agents contraindicated
• In patients with severe renal failure: consider haemodialysis within 2 hours after contrast agent administration – not proven to prevent NSF
### LV Volumes, Function and Mass

#### Male Adults

<table>
<thead>
<tr>
<th></th>
<th>&lt;35 years</th>
<th>≥35 years</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Absolute Values</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>173 ± 29 (115–231)</td>
<td>149 ± 25 (99–199)</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>7 ± 15 (27–87)</td>
<td>43 ± 13 (17–69)</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>118 ± 18 (82–154)</td>
<td>106 ± 19 (68–144)</td>
</tr>
<tr>
<td>EF (%)</td>
<td>67 ± 5 (57–77)</td>
<td>71 ± 6 (59–83)</td>
</tr>
<tr>
<td>Mass (g)</td>
<td>131 ± 21 (89–173)</td>
<td>120 ± 23 (74–166)</td>
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<td><strong>Indexed to BSA</strong></td>
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<tr>
<td>EDV/BSA (ml/m²)</td>
<td>90 ± 11 (68–112)</td>
<td>75 ± 11 (53–97)</td>
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<td>ESV/BSA (ml/m²)</td>
<td>30 ± 7 (16–44)</td>
<td>22 ± 6 (10–34)</td>
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<tr>
<td>SV/BSA (ml/m²)</td>
<td>60 ± 8 (44–76)</td>
<td>53 ± 8 (37–69)</td>
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<tr>
<td>Mass/BSA (g/m²)</td>
<td>67 ± 10 (47–87)</td>
<td>60 ± 9 (42–78)</td>
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</tbody>
</table>

Reference 2). Values are given as mean ± SD; reference ranges in brackets, calculated as ± 2SD of the mean. Analysed with Argus software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.
### LV Volumes, Function and Mass

#### Female Adults

<table>
<thead>
<tr>
<th>Absolute Values</th>
<th>&lt;35 years</th>
<th>≥35 years</th>
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<tbody>
<tr>
<td>EDV (ml)</td>
<td>137 ± 25 (87–187)</td>
<td>128 ± 23 (82–174)</td>
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<tr>
<td>ESV (ml)</td>
<td>43 ± 11 (21–65)</td>
<td>40 ± 12 (16–64)</td>
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<tr>
<td>SV (ml)</td>
<td>96 ± 18 (60–132)</td>
<td>89 ± 16 (57–121)</td>
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<tr>
<td>EF (%)</td>
<td>69 ± 6 (57–81)</td>
<td>69 ± 6 (57–81)</td>
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<tr>
<td>Mass (g)</td>
<td>92 ± 20 (52–132)</td>
<td>92 ± 19 (54–130)</td>
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<table>
<thead>
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<th>≥35 years</th>
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<tr>
<td>EDV/BSA (ml/m²)</td>
<td>80 ± 9 (62–98)</td>
<td>73 ± 11 (51–95)</td>
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<tr>
<td>ESV/BSA (ml/m²)</td>
<td>25 ± 6 (13–37)</td>
<td>23 ± 6 (11–35)</td>
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<tr>
<td>SV/BSA (ml/m²)</td>
<td>55 ± 6 (43–67)</td>
<td>51 ± 8 (35–67)</td>
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<tr>
<td>Mass/BSA (g/m²)</td>
<td>53 ± 9 (35–71)</td>
<td>52 ± 9 (34–70)</td>
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# RV Volumes, Function and Mass

## Male Adults

<table>
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<tbody>
<tr>
<td><strong>Absolute Values</strong></td>
<td></td>
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<tr>
<td>EDV (ml)</td>
<td>203 ± 33 (137–269)</td>
<td>181 ± 28 (125–237)</td>
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<tr>
<td>ESV (ml)</td>
<td>87 ± 20 (47–127)</td>
<td>71 ± 17 (37–105)</td>
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<tr>
<td>SV (ml)</td>
<td>116 ± 19 (78–154)</td>
<td>110 ± 18 (74–146)</td>
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<tr>
<td>EF (%)</td>
<td>57 ± 5 (47–67)</td>
<td>61 ± 6 (49–73)</td>
</tr>
<tr>
<td>Mass (g)</td>
<td>42 ± 8 (26–58)</td>
<td>39 ± 7 (25–53)</td>
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<tbody>
<tr>
<td>EDV/BSA (ml/m²)</td>
<td>104 ± 15 (74–134)</td>
<td>89 ± 11 (67–111)</td>
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<tr>
<td>ESV/BSA (ml/m²)</td>
<td>44 ± 9 (26–62)</td>
<td>34 ± 7 (20–48)</td>
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<tr>
<td>SV/BSA (ml/m²)</td>
<td>59 ± 9 (41–77)</td>
<td>55 ± 8 (39–71)</td>
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<tr>
<td>Mass/BSA (g/m²)</td>
<td>22 ± 4 (14–30)</td>
<td>20 ± 3 (14–26)</td>
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</tbody>
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### Absolute Values

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<tr>
<td>EDV (ml)</td>
<td>152 ± 27 (98–206)</td>
<td>140 ± 37 (66–214)</td>
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<tr>
<td>ESV (ml)</td>
<td>59 ± 12 (35–83)</td>
<td>52 ± 22 (8–96)</td>
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<tr>
<td>SV (ml)</td>
<td>93 ± 17 (59–127)</td>
<td>93 ± 17 (50–126)</td>
</tr>
<tr>
<td>EF (%)</td>
<td>61 ± 3 (55–67)</td>
<td>64 ± 7 (50–78)</td>
</tr>
<tr>
<td>Mass (g)</td>
<td>36 ± 7 (22–50)</td>
<td>33 ± 7 (19–47)</td>
</tr>
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</table>

### Indexed to BSA

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<tr>
<td>EDV/BSA (ml/m²)</td>
<td>89 ± 11 (67–111)</td>
<td>80 ± 19 (42–118)</td>
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<tr>
<td>ESV/BSA (ml/m²)</td>
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<td>30 ± 12 (6–54)</td>
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<td>SV/BSA (ml/m²)</td>
<td>54 ± 7 (40–68)</td>
<td>54 ± 7 (32–68)</td>
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<tr>
<td>Mass/BSA (g/m²)</td>
<td>21 ± 3 (15–27)</td>
<td>19 ± 3 (13–25)</td>
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# Aortic Root Dimensions

## Male

<table>
<thead>
<tr>
<th></th>
<th>20-29 years</th>
<th>30-39 years</th>
<th>40-49 years</th>
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<tbody>
<tr>
<td>Annulus (s)</td>
<td>21.4 ± 2.4</td>
<td>20.7 ± 1.7</td>
<td>21.6 ± 2.0</td>
</tr>
<tr>
<td>Annulus (c)</td>
<td>26.5 ± 1.8</td>
<td>25.2 ± 2.4</td>
<td>25.8 ± 1.5</td>
</tr>
<tr>
<td>Aortic sinus (s)</td>
<td>30.5 ± 3.9</td>
<td>29.8 ± 3.8</td>
<td>32.0 ± 2.4</td>
</tr>
<tr>
<td>Aortic sinus (c)</td>
<td>32.5 ± 3.4</td>
<td>31.8 ± 4.8</td>
<td>33.6 ± 2.6</td>
</tr>
<tr>
<td>Sinotubular junction (s)</td>
<td>23.3 ± 3.4</td>
<td>22.2 ± 4.0</td>
<td>24.4 ± 3.3</td>
</tr>
<tr>
<td>Sinotubular junction (c)</td>
<td>23.7 ± 3.5</td>
<td>22.2 ± 3.0</td>
<td>24.5 ± 2.4</td>
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<tr>
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<th>50-59 years</th>
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<tbody>
<tr>
<td>Annulus (s)</td>
<td>22.8 ± 2.8</td>
<td>23.5 ± 1.8</td>
<td>23.3 ± 2.7</td>
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<tr>
<td>Annulus (c)</td>
<td>26.4 ± 3.7</td>
<td>26.5 ± 1.8</td>
<td>26.6 ± 1.9</td>
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<tr>
<td>Aortic sinus (s)</td>
<td>33.3 ± 6.1</td>
<td>33.6 ± 2.7</td>
<td>35.1 ± 3.7</td>
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<tr>
<td>Aortic sinus (c)</td>
<td>34.7 ± 6.4</td>
<td>35.7 ± 3.3</td>
<td>36.1 ± 3.5</td>
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<tr>
<td>Sinotubular junction (s)</td>
<td>26.6 ± 3.1</td>
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<td>Sinotubular junction (c)</td>
<td>26.5 ± 3.7</td>
<td>27.5 ± 2.4</td>
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Reference 3). Data measured in diastole and presented as mean ± SD in mm. Analyzed from sagittal (s) and coronal (c) SSFP LVOT cines.
# Aortic Root Dimensions

## Female

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<tr>
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<tbody>
<tr>
<td>Annulus (s)</td>
<td>19.5 ± 2.4</td>
<td>19.2 ± 2.3</td>
<td>19.9 ± 2.2</td>
</tr>
<tr>
<td>Annulus (c)</td>
<td>23.6 ± 3.0</td>
<td>22.9 ± 2.3</td>
<td>23.3 ± 1.5</td>
</tr>
<tr>
<td>Aortic sinus (s)</td>
<td>26.5 ± 4.0</td>
<td>26.9 ± 3.1</td>
<td>31.5 ± 2.8</td>
</tr>
<tr>
<td>Aortic sinus (c)</td>
<td>28.5 ± 4.9</td>
<td>28.2 ± 3.1</td>
<td>32.0 ± 2.5</td>
</tr>
<tr>
<td>Sinotubular junction (s)</td>
<td>21.1 ± 3.3</td>
<td>21.8 ± 2.8</td>
<td>25.7 ± 2.3</td>
</tr>
<tr>
<td>Sinotubular junction (c)</td>
<td>21.5 ± 2.7</td>
<td>22.1 ± 2.7</td>
<td>25.5 ± 2.1</td>
</tr>
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<tr>
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<td>29.1 ± 2.5</td>
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</tr>
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<td>Sinotubular junction (c)</td>
<td>23.4 ± 2.1</td>
<td>24.7 ± 1.6</td>
<td>25.1 ± 1.3</td>
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</table>

Reference 3). Data measured in diastole and presented as mean ± SD in mm. Analyzed from sagittal (s) and coronal (c) SSFP LVOT cines.

Index
<table>
<thead>
<tr>
<th>Acceleration technique</th>
<th>Comment</th>
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<tbody>
<tr>
<td>Reduce <strong>number of slices</strong> acquired per breath-hold</td>
<td>Increases overall scan time</td>
</tr>
<tr>
<td>Reduce <strong>number of phases</strong> for each breath-hold:</td>
<td>Prone to artefacts</td>
</tr>
<tr>
<td>- by reducing <strong>acquisition matrix</strong> (scan or phase percentage)</td>
<td>Increases overall scan time</td>
</tr>
<tr>
<td>- by reducing <strong>FOV</strong></td>
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<tr>
<td>Increase <strong>voxel size</strong></td>
<td>Decreases spatial resolution</td>
</tr>
<tr>
<td>Use <strong>parallel imaging</strong></td>
<td></td>
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<tr>
<td>Use <strong>respiratory navigator</strong></td>
<td></td>
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<tr>
<td>Acquire images in <strong>inspiration</strong></td>
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<tr>
<td><strong>Consider general anaesthesia</strong></td>
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<tr>
<td><strong>Acquire images in inspiration</strong></td>
<td></td>
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<tr>
<td><strong>Varying slice position with each breath-hold</strong></td>
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**Index**
<table>
<thead>
<tr>
<th>Technique</th>
<th>Comment</th>
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<tbody>
<tr>
<td>Heart rate and/or rhythm control</td>
<td>Use beta-blockers or other antiarrhythmic medication</td>
</tr>
<tr>
<td>Use Arrhythmia Rejection</td>
<td>Increases breath-hold time</td>
</tr>
<tr>
<td>Use Prospective triggering</td>
<td>Reduces SNR</td>
</tr>
<tr>
<td>Use Real-time imaging</td>
<td>Reduces temporal and spatial resolution as well as SNR</td>
</tr>
</tbody>
</table>
### Anatomy Module

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

### LV function Module

1. **Cine SSFP** pulse sequence (parallel imaging as required)
2. 2-ch, 4-ch, 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 ≤ 45ms

### RV function Module

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 module
   - Slice thickness 6-8mm, inter-slice gap 0mm
3. Temporal resolution ≤45ms
Tips & Tricks (Anatomy Module)

1. Scan in diastole to reduce motion artefacts

Tips & Tricks (LV / RV Function Module)

1. To reduce breath-hold times use acceleration techniques
2. Contouring:
   - In a healthy heart there is usually one less slice to contour in end-systole at the base of the heart (longitudinal LV shortening). Correlate SA to long axis view if available to identify mitral 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.
3. RV volumes are more reproducible when calculated from an axial imaging plane.
1. **Scout imaging** as per LV function module

2. **Saturation-recovery gradient echo pulse sequence** (GRE, gradient echo-echo planar (GRE-EPI), or SSFP readout)

3. **Parallel imaging** (twofold acceleration, if available)

4. **SA view imaging** (at least three slices per heartbeat);
   - 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. **“Dummy” scan** to check
   - Correct slice positioning
   - Artefacts
   - ECG triggering at every single heartbeat

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**

![Diagram showing slice positioning](image)
1. 2D-segmented IR GRE imaging during diastolic rest period
2. 4-ch, 3-ch, 2-ch, SA images
3. In-plane resolution : <2mm
4. EGE: image 1-3min after contrast, TI >400ms
5. LGE: ≥10min after Gd injection (0.1 – 0.2mmol /kg)
   • The delay may be shorter if lower Gd doses are used
   • The delay may be increased 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 couple of minutes,** because the correct TI for “nulling” of normal myocardium slowly 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
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 VENC**:  
   - Normal systemic flow: 150 cm/s  
   - Normal right-sided flow: 100 cm/s  
   - Adjust in pathological situations (severe valve stenosis > 400 cm/s)

5. Choose **adequate spatial resolution**  
   - minimum of 4-6 pixels per vessel diameter

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*Volume time curve from flow velocity encoding through the ascending aorta in a patient with severe aortic regurgitation*
1. **VENC settings:**
   - Optimal within 25% of the true peak velocity
     - Too low: flow aliasing
     - Too high: underestimating velocity
   - Correct direction of flow (R-L, F-H)
   - Image plane distal from valve leaflet tips
   - Flow assessment: perpendicular to the vessel
   - Max. 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

*Sagittal (A) and coronal (B) slice positioning for aortic stenosis*
Edema Module

1. **T2w imaging**
2. **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**
5. **Use body coil** or alternatively **functional surface coil** intensity correction algorithms to correct for coli-related signal differences
6. **Slow flow artefacts** may cause high signal at endocardial border

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*Myocardial infarction with inferior edema on T2w images (A) and LGE (B)*
1. Prepare **infusion pump** with contrast agent and flush
   Gd 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
1. **Determine coronary rest period**
   - Acquire HLA with **high temporal resolution** (50 phases)

2. **Navigator-gated, free-breathing 3D pulse sequence:**
   - **Trans-axial slices** (from the proximal main pulmonary artery to the middle of the right atrium; entire cardiac coverage if desired).
   - **Slice thickness**: 1-1.5 mm
   - **Spatial resolution in-plane**: 1.0 mm or less
   - **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 hemi-diaphragm

3. **Optional:**
   - Consider contrast to increase vessel conspicuity
   - Breath-hold techniques if poor image quality or if navigators are unavailable or are of poor quality
   - T2-prepared sequence may be useful
Tips & Tricks

1. **Problems identifying coronary rest period:**
   - repeat high temporal resolution 4-ch scan at the correct HR
   - **Consider cine scan during free-breathing** if HR changes significantly during breath-hold
   - **Check during systole** with a tight window (<50 ms)
   - As a compromise, scan **with longest trigger delay and a tight window** (<50 ms)

2. Coronary rest period may differ between LCA and RCA

3. **High HR** (≥ 90bpm): Use shortest scan window possible to minimize blurring

4. **Keep scan times to a sensible limit**

5. Higher spatial resolution equals longer scan times

4-ch view showing the RCA in diastole
1. **Scout imaging** as per LV function module
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*
T2★ Module

1. T2★ quantitation is a standard CMR technique for **disease monitoring** and **guiding chelation therapy** in cardiac iron-loading conditions

2. **Single breath-hold, multi-echo, T2★ sequence** (gradient echo or modified black blood sequence)

3. **Single mid-ventricular slice**

4. **Single transaxial slice of the liver**

**Tips & Tricks**

1. **Ensure good** patient breath-holding for the heart and the liver scans 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)**
### Artefacts

#### Wrapping artefact (fold-over, back-folding)
- Increase FOV
- Add phase encoding (phase-oversampling, foldover suppression, no phase wrap)
- Swap phase and frequency direction
- Use selective tissue saturation bands
- Use a surface coil

#### 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 (gradient moment nulling, gradient motion rephasing)
- 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 bSSFP acquisition

## Chemical shift artefact
- Compare with other images as they are sequence dependent

## Dark rim artefact
- Often seen in perfusion imaging
- Reduce contrast dose/infusion speed
- Increase in-plane spatial resolution
Artefacts

Radiofrequency interference artefact

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

Slow flow artefact

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

Metallic artefact

• Usually less prominent on spin echo images than gradient echo images
Ischemic Heart Disease

Perfusion

Protocol

1. **Anatomy** module
2. **Myocardial perfusion** module “dummy“
3. **Myocardial perfusion** module STRESS
4. **LV function** module
5. **Myocardial perfusion** module REST
6. **LGE** module

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 inducible **perfusion defect**
4. **Correlation** between **scar and perfusion defect**
5. Comment on **suitability of revascularization** based on ischemia and viable myocardium
6. (Presence and location of **artefacts**)

Index
Key Issues

1. Check BP /monitor ECG during adenosine perfusion

2. Adenosine dose:
   • 140mcg/kg/min
   • Consider 170 or 210mcg/kg/min, if hemodynamic response is inadequate or after caffeine intake

3. Contraindications for adenosine: known hypersensitivity, 2nd /3rd AV nodal block, severe reversible airways disease

Tips & Tricks

1. One i.v. cannula (Y connector) for Adenosine and Gd is safe

2. Use “3 out of 5” technique to position perfusion slices

3. Note: Segment 17 is not visualized on 3 slice SA perfusion scan
Ischemic Heart Disease
Wall Motion

Protocol

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

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 (=ischemia)
2. Presence and transmural extent of **scar**
3. Summarize: **resting function, contractile reserve, wall motion index, ischemia 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 dobutamine**: narrow-angle glaucoma, myasthenia gravis, obstructive uropathy, obstructive gastrointestinal disorders

Tips & Tricks

1. Use “3 out of 5” technique (perfusion module) to position SA slices
Ischemic Heart Disease

Wall Motion

<table>
<thead>
<tr>
<th>Ischemic Conditions</th>
<th>Myocardial Wall Motion</th>
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<tbody>
<tr>
<td></td>
<td>Rest</td>
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<tr>
<td>Normal</td>
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<tr>
<td>Ischemia</td>
<td><img src="image" alt="Ischemia Rest" /></td>
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<tr>
<td>Hibernation</td>
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<tr>
<td>Subendocardial Scar</td>
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</tr>
<tr>
<td>Transmural scar</td>
<td><img src="image" alt="Transmural scar Rest" /></td>
</tr>
</tbody>
</table>

Wall Motion Score Index (WMSI)

(sum of wall motion scores / number of segments)

| Normal      | 1 |
| Hypokinetic | 2 |
| Akinetic    | 3 |
| Dyskinetic  | 4 |
| Aneurysmal  | 5 |

A WMSI of 1 is considered normal
17 Segment Model

Modified from reference 4)
Ischemic Heart Disease

Coronary Supply

4 chamber

2 chamber

3 chamber

basal

mid

apical

LAD

LAD or CX

CX

LAD or RCA

RCA

CX or RCA

Modified from reference 4)
Ischemic Heart Disease

Acute Myocardial Infarction

Protocol

1. **Anatomy** module
2. **LV function** module
3. **Edema** module
4. **EGE – LGE** module

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 edema** (=area at risk)
3. Presence and transmural extent of **scar**
4. Presence and extend of **microvascular obstruction** (MVO)

*Acute myocardial infarction: A) inferoseptal edema on T2w SA B, C) MVO on 4-ch EGE, LGE*
Key Points

1. T2w imaging may differentiate acute from chronic myocardial infarction

2. Microvascular obstruction:
   - 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
Dilated Cardiomyopathy

Protocol

1. **Anatomy** module
2. **LV function** module
3. **Edema** module
4. **RV function** module
5. **LGE** module

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 end extent of mid-wall fibrosis

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)
Hypertrophic Cardiomyopathy

Protocol

1. **Anatomy** module
2. **LV function** module
3. **LVOT** cines (2 orthogonal views)
4. **Velocity encoding** module in- and through- LVOT planes
5. **LV tagging** (3 SA slices, 4ch) - optional
6. **LGE** module

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**

**HCM: Septal hypertrophy and SAM of anterior mitral leaflet on SA (A) and 3-ch (B)**
Key Points

1. **Risk factors for sudden cardiac death in HCM:**
   - Positive family history
   - Syncope
   - Frequent repetitive NSVT
   - Blood pressure drop during exercise
   - Massive LV hypertrophy ≥30mm
   - Presence and extent of LGE
2. **Consider possible obstruction under stress conditions**

Tips & Tricks

1. **LGE at the insertion points** of the RV to the LV are non-specific and often seen even in normal subjects
2. **Suggestive for HCM:**
   - Localized hypertrophy
   - Reduced contraction of hypertrophied segments
   - Presence of LGE
3. **Tagging** may help identify wall motion abnormalities
Left Ventricular Non-Compaction Cardiomyopathy

Protocol

1. **Anatomy** module
2. **LV function** module
3. **LGE** module

Report

1. **Dimensions, mass** (corrected for BSA) and **function**
   - LV: EDV, ESV, SV, EF
   - Mass of non-compacted and compacted layer
2. **Regional wall motion abnormalities**
3. Location and extent of segments with *increased non-compacted to compacted (NC/C) myocardial ratio*

**IVNC:** Significant non-compacted myocardial layer, primarily in the lateral wall on SA (A) and 4-ch (B)
Key Points

1. **Current diagnostic criteria:**
   - NC/C $\geq 2.3 : 1$ on end-diastolic image*
     - Note: NC/C 2:1 on end-systolic echo images
   - Non-compacted LV mass above 20% of the global mass

2. **LGE** may represent severe or late forms of LVNC

3. **Diagnosis may not be based on imaging criteria alone**
   - Often over-diagnosed, particular in DCM (thin compacted myocardium) and in patients of Afro-American descent
   - Current diagnostic criteria may overdiagnose LVNC and new guidance is anticipated

Tips & Tricks

1. Consider associated congenital defects (Ebstein anomaly, coarctation of the aorta, bicuspid aortic valve...)

*Measured on 4-ch, 3-ch and 2-ch long axis cines
Protocol

1. **Anatomy** module
2. **LV function** module
3. **RV function** module (axial and RVOT)
   - Slice thickness 6-8mm without inter-slice gap
4. T1w axial **black blood** images (optional)
5. T1w axial **fat suppressed black blood** images (optional)
6. **LGE** module 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** (if acquired)
4. Presence and extent of **fibrosis**
Key Points

1. Diagnosis cannot be based on imaging criteria alone
   - See modified 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

**ARVC: Dilated, aneurysmatic RV with LG enhancement on 4-ch (A), LGE (B)**
### 1. Global or regional dysfunction and structural alterations

**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

| Major | Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
|       | and 1 of the following:
|       | - Ratio of RV EDV to BSA ≥110mL/m² (male) or ≥100mL/m² (female)
|       | - or RV ejection fraction ≤40%

| Minor | Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
|       | and 1 of the following:
|       | - Ratio of RV EDV to BSA ≥100 to <110mL/m² (male) or ≥90 to <100mL/m² (female)
|       | - or RV EF >40% to ≤45% |

### 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 ≥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 ≥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 ≥120ms)

**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 ≥1 of 3 parameters in the absence of a QRS duration of ≥110ms on the standard ECG
  • Filtered QRS duration (fQRS) ≥114ms
  • Duration of terminal QRS <40µV (low-amplitude signal duration) ≥38ms
  • Root-mean-square voltage of terminal 40ms ≤20 µV
  • Terminal activation duration of QRS ≥55ms 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† 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

Reference 5)
Protocol

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

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 **edema**
3. Presence and location of **LGE**
4. **Pericardial effusion / enhancement**

SA views show edema on T2w (A), mid-wall (B) and pericardial enhancement on LGE (C).
Key Points

1. **Diagnostic CMR criteria**
   - Myocardial inflammation (≥ 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 ≥2.0
     - Global myocardial SI increase on EGE
       - SI ratio of myocardium over skeletal muscle of ≥ 4.0
       - or absolute myocardial enhancement of ≥45%
     - At least 1 focal lesion with non-ischemic 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

*Modified from reference 7*)
Protocol

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

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

Cardiac amyloidosis: Hypertrophic LV on 4-ch (A) with diffuse sub-endocardial LGE (B)
Key Points

1. **Restrictive LV pattern** (non-dilated ventricles, preserved LV function, restrictive filling pattern, enlarged LA / RA) and global LV hypertrophy

2. **LV hypertrophy**

3. Consider amyloidosis if myocardial nulling difficult to achieve on LGE images despite good technique

4. **Abnormal** myocardial and blood-pool gadolinium **kinetics**
   - Faster Gd washout from blood and myocardium

5. **Epicardial - endocardial gradient** on early imaging

6. **LGE pattern:**
   - Predominantly global sub-endocardial distribution

7. **Atrial septum hypertrophy of >6mm** (in <20% of cases)

8. **Pericardial** and **pleural effusion** are common

9. Cardiac involvement without hyperenhancement is rare

Tips & Tricks

1. Consider T1 mapping techniques for the detection of global gadolinium uptake

2. CMR guidance for myocardial biopsy
Sarcoidosis

Protocol

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

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**

Cardiac sarcoidosis: typical granulomas in 2-ch (A) and SA (B)
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 edema** 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
Endomyocardial Fibrosis

Protocol

1. **Anatomy** module
2. **LV function** (RV function) module
3. **Edema** module
4. **EGE / LGE** module

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**

A

![Hypertrophied LV (A) with endocardial fibrosis on LGE (B)](image)

B
### 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

### Tips & Tricks

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

Protocol

1. **Anatomy** and **LV function** (RV function) module
2. **T2★ module**

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★ values of heart and liver**

Key Points

1. **Restrictive LV pattern** (non-dilated ventricles, preserved LV function, restrictive filling pattern, enlarged LA / RA)
2. **Diagnostic signs:**
   - LV dysfunction; LV hypertrophy
   - Focal signal loss in native T1- and T2-weighted images
   - Abnormally "dark" liver
2. **Diagnostic T2★ values:**
   - Septal myocardium <20ms; liver tissue ≤6.3ms
3. **Follow-up** of iron loading to guide **chelation therapy**
4. Single cardiac or liver involvement is possible

Tips & Tricks

1. Assess **T2★ values in the septum (less artefacts)**
## Protocol

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

## 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 **edema**
3. Presence of **LGE**

*Typical Tako-Tsubo pattern of apical ballooning during systole*
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. Edema 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**
## Protocol

1. **Anatomy** module including **T1 and T2 weighting**
2. **LV function** module
3. **Consider:**
   - Tumor module
   - Valve module
   - Real-time free-breathing cine (2 planes)
4. **LGE** module

## 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**
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, myxedema, neoplastic
   - Regional: postoperative, trauma, purulent, cyst

### Tips & Tricks

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

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<th>Cine</th>
<th>SI (b-SSFP)</th>
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<td>simple</td>
<td>↑</td>
</tr>
<tr>
<td>Exudate</td>
<td>↓↑</td>
<td>complex</td>
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<td>Chylous</td>
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<td>simple</td>
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</table>
Constrictive Pericarditis

Protocol

1. **Anatomy** module including T1w and T2w
2. **LV / RV function** module
3. **RV function** module (axial and RVOT)
4. **Real-time dynamic respiratory cine**
5. **LGE** module

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 ≥3mm**
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
Protocol

1. Coronary Artery Imaging Module

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, ...

3. Anomalies of intrinsic coronary arterial anatomy
   • Ectasia, aneurysm, hypoplasia, ...
   • Intramural coronary artery (muscular bridge)

4. Anomalies of coronary termination

5. Anomalous collateral vessels
Anomalous Coronary Arteries

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 ischemia:**
   - 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 to demonstrate a regional wall motion abnormality (if inter-arterial course)

---

Left coronary artery arising from the right coronary cusp with a retro-aortal course (A). Normal origin of the RCA (B)
Aortic Disease

Protocol

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

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 btw. left carotid and subclavian a.
   • Desc Ao at level of PA and diaphragm
3. Aortic position (left or right) and tortuosity
4. Atherosclerosis, aneurysm, dissection, inflammation
5. Aortic flow
6. Associated aortic valvular stenosis or regurgitation
## Key Points

1. Method of choice for **non-acute aortic diseases**

2. **Standardize protocol:**
   - Measure in **end-diastole** from **cine imaging**, if possible
   - Use **same slice thickness** (<7mm)
   - **Aortic root** (from 2 orthogonal LVOT cines or AV stack)
   - **Asc / desc Ao** (from sagittal oblique aorta cines or alternatively from MRA, if necessary)

## Tips & Tricks

1. Always perform **arterial and venous MRA**

2. **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
   - Angeled view of aorta, if taken from transaxial stack
   - Inclusion of aortic wall, if taken from BB images
Valvular Heart Disease

Protocol

1. Anatomy / LV function / RV function module
2. Optimized cine views:
   • Slice thickness 5mm
   • Two orthogonal cine stacks through the valve
   • One cine stack parallel to the annulus
3. Phase contrast velocity encoded module

Report

1. Dimensions, mass (corrected for BSA) and function
   • LV: EDV, ESV, SV, EF, mass
   • RV: EDV, ESV, SV, EF
2. Valve morphology: leaflets, annulus, chordae
3. Valve stenosis
   • Mean / peak valvular gradients
   • Minimum valve area
4. Valve regurgitation
   • Regurgitation volume and fraction
   • Estimated orifice area
1. **CMR** is a reasonable alternative if poor echocardiographic image quality (lower spatial and temporal resolution)

2. **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

3. **VENC** settings (see “Flow velocity encoding” section)

<table>
<thead>
<tr>
<th><strong>Pulse sequence</strong></th>
<th><strong>Indication</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>SSFP cine</td>
<td>• Anatomy and motion</td>
</tr>
<tr>
<td></td>
<td>• LV / RV volumes and function</td>
</tr>
<tr>
<td>Gradient echo cine</td>
<td>• Valve leaflet motion</td>
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<tr>
<td></td>
<td>• Turbulent flow</td>
</tr>
<tr>
<td>Flow velocity encoding</td>
<td>• Forward / regurgitant volume</td>
</tr>
</tbody>
</table>
## Calculation of regurgitant volume in SINGLE valve disease

**Aortic regurgitation**
- Regurgitation volume/fraction from phase contrast VENC above aortic valve
- Alternatively LV SV – RV SV

**Mitral regurgitation**
- SV from phase contrast VENC above aortic valve – LV SV
- Alternatively LV SV – RV SV

**Pulmonary regurgitation**
- Regurgitation volume/fraction from phase contrast VENC above pulmonary valve
- RV SV – LV SV

**Tricuspid regurgitation**
- SV from phase contrast VENC above pulmonary valve – LV RV
- Alternatively RV SV – LV SV

## Limitations

1. **Degree of stenosis or regurgitation – cines imaging**
   - Visual assessment from cine images alone is NOT recommended due to a signal void in turbulent flow

2. **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
Limitations

3. **Flow velocity encoding— forward flow / peak velocity**
   - VENC tends to underestimate velocities due to
     - Partial volume averaging
     - Slice orientation NOT perpendicular to the flow

4. **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

Tips & Tricks

1. Reduce slice thickness to <6mm
2. Consider overlapping of slices
3. Patchy mid-wall fibrosis in conjunction with LV hypertrophy is a prognostic sign in aortic stenosis
4. Aortic regurgitation fraction of >33% predicts symptom development and the need for valve replacement
5. A pulmonary regurgitation fraction of >40% predicts symptom development and the need for valve replacement
Cardiac Masses

Protocol

1. High resolution anatomy module
2. Cine imaging in all standard and targeted planes
3. In 2 optimized orthogonal planes
   • T1w black blood images with fat suppression
   • T1w black blood images pre and post contrast
   • T2w
   • First pass myocardial perfusion imaging
   • EGE and LGE

Report

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

Key Points

1. Cardiac metastatic lesions are up to 1000 times more common than primary tumors

2. Common sources of metastatic lesions
   • Melanoma, thyroid cancer, breast cancer, renal carcinoma, soft tissue carcinoma, lung cancer, esophageal cancer, hepatocellular carcinoma

3. Common benign primary tumors (70%)
   • Myxoma, lipoma, fibroelastoma, fibroma, rhabdomyoma, hemangioma

4. Common malignant primary tumors (30%)
   • Angiosarcoma, rhabdomyosarcoma, mesothelioma, fibrosarcoma, lymphoma

5. Consider pseudotumors:
   • normal heart structures, thrombus, cyst or vegetation

Tips & Tricks

1. Very small and highly mobile masses (e.g. vegetation, fibroelastoma) might be missed with CMR

2. CMR allows tissue characterisation, but cannot provide histopathologic information.
# Cardiac Masses Tissues Characteristics

<table>
<thead>
<tr>
<th>Cardiac Mass</th>
<th>T1w*</th>
<th>T2w*</th>
<th>LGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pseudotumors</strong></td>
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</tr>
<tr>
<td>Thrombus</td>
<td>Low</td>
<td>Low</td>
<td>No uptake†</td>
</tr>
<tr>
<td></td>
<td>(high if recent)</td>
<td>(high if recent)</td>
<td></td>
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<tr>
<td>Pericardial cyst</td>
<td>Low</td>
<td>High</td>
<td>No uptake</td>
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<tr>
<td><strong>Benign</strong></td>
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<tr>
<td>Myxoma</td>
<td>Isointense</td>
<td>High</td>
<td>Heterogeneous</td>
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<tr>
<td>Lipoma</td>
<td>High‡</td>
<td>High‡</td>
<td>No uptake</td>
</tr>
<tr>
<td>Fibroma</td>
<td>Isointense</td>
<td>Low</td>
<td>Hyperenhanced</td>
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<tr>
<td>Rhabdomyoma</td>
<td>Isointense</td>
<td>Isointense/high</td>
<td>No/min. uptake</td>
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<tr>
<td><strong>Malignant</strong></td>
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<tr>
<td>Angiosarcoma</td>
<td>Heterogenous</td>
<td>Heterogenous</td>
<td>Heterogeneous</td>
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<tr>
<td>Rhabdomyosarcoma</td>
<td>Isointense</td>
<td>Hyperintense</td>
<td>Homogeneous</td>
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<td>Undifferentiated sarcoma</td>
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<td>Hyperintense</td>
<td>Heterogeneous/V variable</td>
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<td>Lymphoma</td>
<td>Isointense</td>
<td>Isointense</td>
<td>No/min. uptake</td>
</tr>
<tr>
<td>Metastasis §</td>
<td>Low</td>
<td>High</td>
<td>Heterogeneous</td>
</tr>
</tbody>
</table>

Modified from reference 7). * 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.
<table>
<thead>
<tr>
<th>Sequence Type</th>
<th>GE</th>
<th>Philips</th>
<th>Siemens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast Spin Echo</td>
<td>FSE (Fast SE)</td>
<td>TSE (Turbo SE)</td>
<td>Turbo SE</td>
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<td>Gradient recalled echo</td>
<td>GRE</td>
<td>FFE</td>
<td>GRE</td>
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<td>Spoiled gradient echo</td>
<td>SPGR / MPSPGR</td>
<td>T1 FFE</td>
<td>FLASH</td>
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<td>Balanced gradient echo</td>
<td>FIESTA</td>
<td>bFFE / bTFE</td>
<td>TrueFISP</td>
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<td>Gradient echo – echo planar</td>
<td>GRE EPI</td>
<td>FFE-EPI / TFE-EPI</td>
<td>EPIFI</td>
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<td>Contrast enhanced MRA</td>
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<td>Bolus Trak</td>
<td>Care Bolus</td>
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<td>k-space lines</td>
<td>Views per segment</td>
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<td>No of segments</td>
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<td>Parallel imaging: Image-based reconstruction</td>
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</tr>
</tbody>
</table>


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