

Cardiovascular Magnetic Resonance

Pocket Guide

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CMR Pocket Guide

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Foreword

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.

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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 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

Reference 1)

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Safety

Nephrogenic Systemic Fibrosis

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

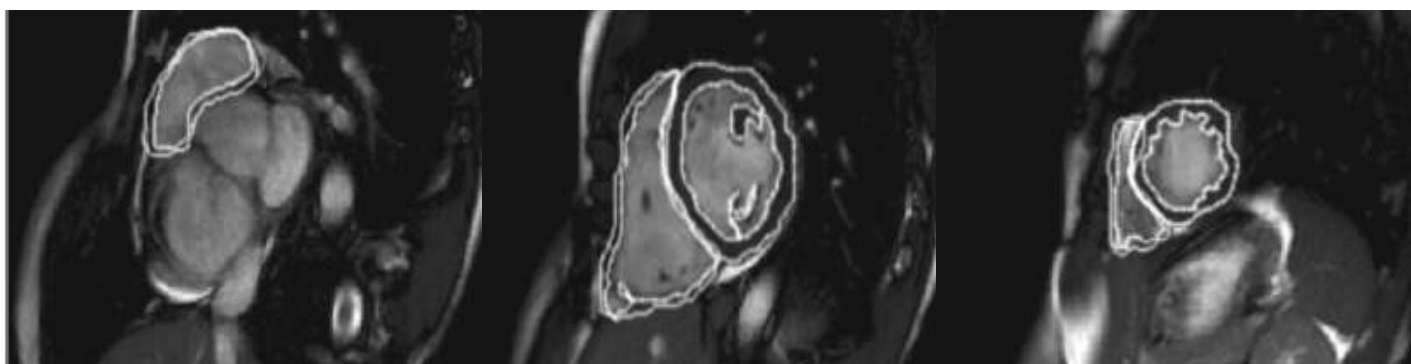
Tips & Tricks

- **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

Absolute Values	<35 years	≥35 years
EDV (ml)	173 ± 29 (115–231)	149 ± 25 (99–199)
ESV (ml)	7 ± 15 (27–87)	43 ± 13 (17–69)
SV (ml)	118 ± 18 (82–154)	106 ± 19 (68–144)
EF (%)	67 ± 5 (57–77)	71 ± 6 (59–83)
Mass (g)	131 ± 21 (89–173)	120 ± 23 (74–166)

Indexed to BSA	<35 years	≥35 years
EDV/BSA (ml/m ²)	90 ± 11 (68–112)	75 ± 11 (53–97)
ESV/BSA (ml/m ²)	30 ± 7 (16–44)	22 ± 6 (10–34)
SV/BSA (ml/m ²)	60 ± 8 (44–76)	53 ± 8 (37–69)
Mass/BSA (g/m ²)	67 ± 10 (47–87)	60 ± 9 (42–78)



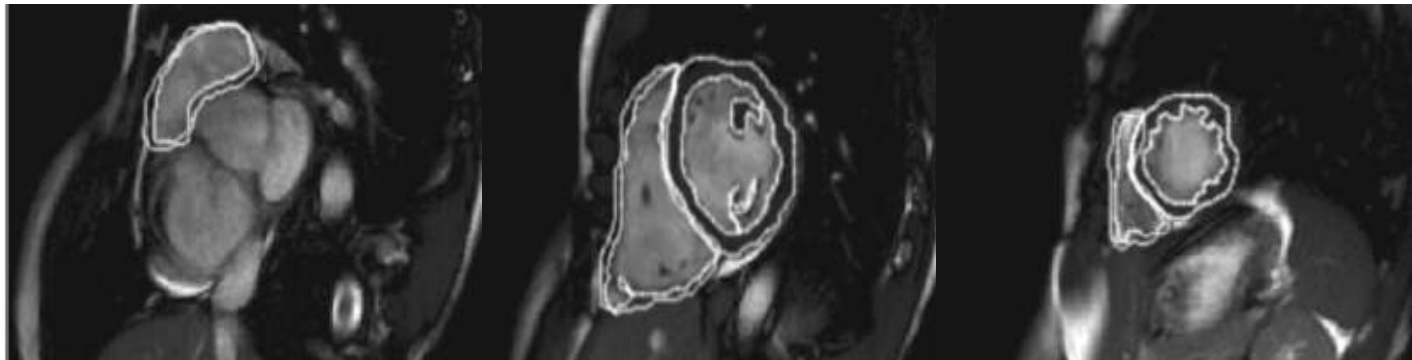
Reference 2). Values are given as mean ± SD; reference ranges in brackets, calculated as ± 2SD of the mean. Analysed with Argus software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.

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LV Volumes, Function and Mass Female Adults

Absolute Values	<35 years	≥35 years
EDV (ml)	137 ± 25 (87–187)	128 ± 23 (82–174)
ESV (ml)	43 ± 11 (21–65)	40 ± 12 (16–64)
SV (ml)	96 ± 18 (60–132)	89 ± 16 (57–121)
EF (%)	69 ± 6 (57–81)	69 ± 6 (57–81)
Mass (g)	92 ± 20 (52–132)	92 ± 19 (54–130)

Indexed to BSA	<35 years	≥35 years
EDV/BSA (ml/m ²)	80 ± 9 (62–98)	73 ± 11 (51–95)
ESV/BSA (ml/m ²)	25 ± 6 (13–37)	23 ± 6 (11–35)
SV/BSA (ml/m ²)	55 ± 6 (43–67)	51 ± 8 (35–67)
Mass/BSA (g/m ²)	53 ± 9 (35–71)	52 ± 9 (34–70)

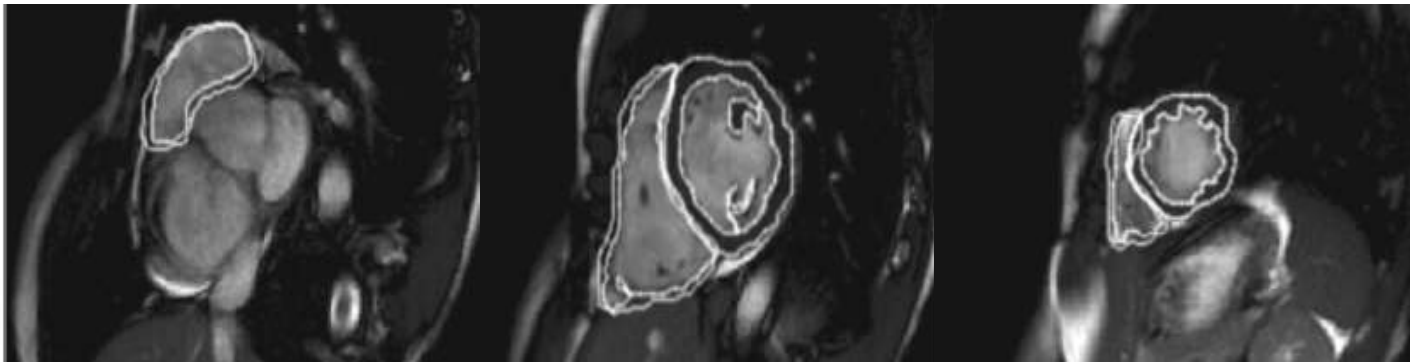


Reference 2). Values are given as mean ± SD; reference ranges in brackets, calculated as ± 2SD of the mean. Analysed with Argus software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.

RV Volumes, Function and Mass Male Adults

Absolute Values	<35 years	≥35 years
EDV (ml)	203 ± 33 (137–269)	181 ± 28 (125–237)
ESV (ml)	87 ± 20 (47–127)	71 ± 17 (37–105)
SV (ml)	116 ± 19 (78–154)	110 ± 18 (74–146)
EF (%)	57 ± 5 (47–67)	61 ± 6 (49–73)
Mass (g)	42 ± 8 (26–58)	39 ± 7 (25–53)

Indexed to BSA	<35 years	≥35 years
EDV/BSA (ml/m ²)	104 ± 15 (74–134)	89 ± 11 (67–111)
ESV/BSA (ml/m ²)	44 ± 9 (26–62)	34 ± 7 (20–48)
SV/BSA (ml/m ²)	59 ± 9 (41–77)	55 ± 8 (39–71)
Mass/BSA (g/m ²)	22 ± 4 (14–30)	20 ± 3 (14–26)

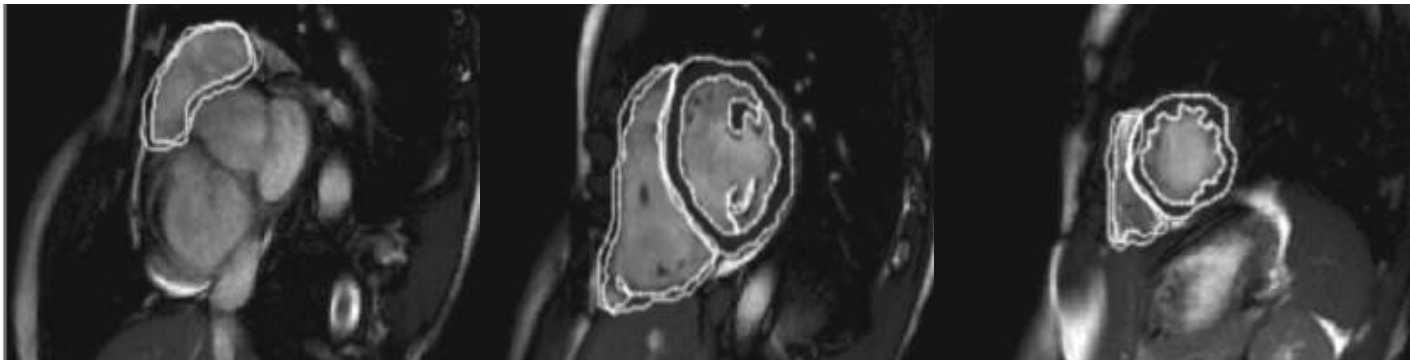


Reference 2). Values are given as mean ± SD; reference ranges in brackets, calculated as ± 2SD of the mean. Analysed with Argus software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.

RV Volumes, Function and Mass Female Adults

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

Indexed to BSA	<35 years	≥35 years
EDV/BSA (ml/m ²)	89 ± 11 (67–111)	80 ± 19 (42–118)
ESV/BSA (ml/m ²)	35 ± 5 (25–45)	30 ± 12 (6–54)
SV/BSA (ml/m ²)	54 ± 7 (40–68)	54 ± 7 (32–68)
Mass/BSA (g/m ²)	21 ± 3 (15–27)	19 ± 3 (13–25)

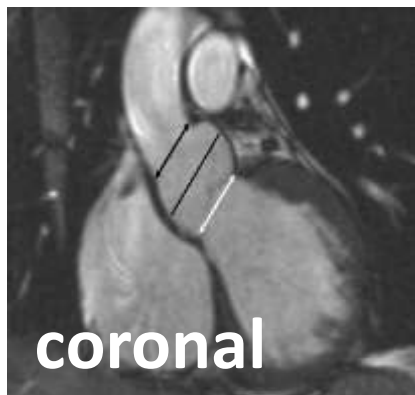


Reference 2). Values are given as mean ± SD; reference ranges in brackets, calculated as ± 2SD of the mean. Analysed with Argus software from short axis SSFP cine images. These values may vary depending on image sequence, acquisition technique and contouring.

Aortic Root Dimensions

Male

	20-29 years	30-39 years	40-49 years
Annulus (s)	21.4 ± 2.4	20.7 ± 1.7	21.6 ± 2.0
Annulus (c)	26.5 ± 1.8	25.2 ± 2.4	25.8 ± 1.5
Aortic sinus (s)	30.5 ± 3.9	29.8 ± 3.8	32.0 ± 2.4
Aortic sinus (c)	32.5 ± 3.4	31.8 ± 4.8	33.6 ± 2.6
Sinotubular junction (s)	23.3 ± 3.4	22.2 ± 4.0	24.4 ± 3.3
Sinotubular junction (c)	23.7 ± 3.5	22.2 ± 3.0	24.5 ± 2.4
	50-59 years	60-69 years	70-79 years
Annulus (s)	22.8 ± 2.8	23.5 ± 1.8	23.3 ± 2.7
Annulus (c)	26.4 ± 3.7	26.5 ± 1.8	26.6 ± 1.9
Aortic sinus (s)	33.3 ± 6.1	33.6 ± 2.7	35.1 ± 3.7
Aortic sinus (c)	34.7 ± 6.4	35.7 ± 3.3	36.1 ± 3.5
Sinotubular junction (s)	26.6 ± 3.1	27.6 ± 3.6	28.3 ± 2.7
Sinotubular junction (c)	26.5 ± 3.7	27.5 ± 2.4	27.8 ± 1.7



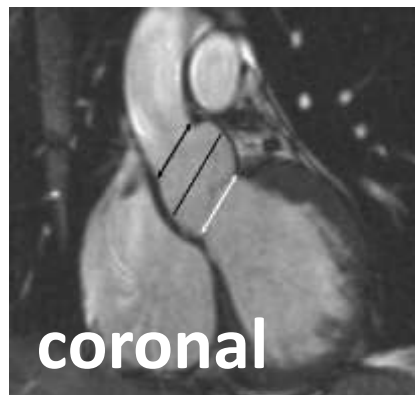
Reference 3). Data measured in diastole and presented as mean ± SD in mm. Analyzed from sagittal (s) and coronal (c) SSFP LVOT cines

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Aortic Root Dimensions

Female

	20-29 years	30-39 years	40-49 years
Annulus (s)	19.5 ± 2.4	19.2 ± 2.3	19.9 ± 2.2
Annulus (c)	23.6 ± 3.0	22.9 ± 2.3	23.3 ± 1.5
Aortic sinus (s)	26.5 ± 4.0	26.9 ± 3.1	31.5 ± 2.8
Aortic sinus (c)	28.5 ± 4.9	28.2 ± 3.1	32.0 ± 2.5
Sinotubular junction (s)	21.1 ± 3.3	21.8 ± 2.8	25.7 ± 2.3
Sinotubular junction (c)	21.5 ± 2.7	22.1 ± 2.7	25.5 ± 2.1
	50-59 years	60-69 years	70-79 years
Annulus (s)	20.1 ± 1.9	20.4 ± 1.1	20.2 ± 1.5
Annulus (c)	22.7 ± 2.1	22.3 ± 1.5	23.3 ± 1.5
Aortic sinus (s)	29.1 ± 2.5	30.1 ± 2.5	30.2 ± 2.0
Aortic sinus (c)	30.2 ± 2.3	31.0 ± 2.7	31.3 ± 1.8
Sinotubular junction (s)	24.1 ± 1.9	25.1 ± 3.0	25.0 ± 2.0
Sinotubular junction (c)	23.4 ± 2.1	24.7 ± 1.6	25.1 ± 1.3



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

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Imaging

Poor Breath-Holders

Acceleration technique	Comment
<ul style="list-style-type: none">• Reduce number of slices acquired per breath-hold	<ul style="list-style-type: none">• Increases overall scan time
<ul style="list-style-type: none">• Reduce number of phases for each breath-hold:<ul style="list-style-type: none">- by reducing acquisition matrix (scan or phase percentage)- by reducing FOV	<ul style="list-style-type: none">• Reduces SNR• Increases spatial resolution
<ul style="list-style-type: none">• Increase voxel size	<ul style="list-style-type: none">• Decreases spatial resolution
<ul style="list-style-type: none">• Use parallel imaging	<ul style="list-style-type: none">• Prone to artefacts
<ul style="list-style-type: none">• Use respiratory navigator	<ul style="list-style-type: none">• Increases overall scan time
<ul style="list-style-type: none">• Acquire images in inspiration	<ul style="list-style-type: none">• Varying slice position with each breath-hold
<ul style="list-style-type: none">• Consider general anaesthesia	

Imaging

Patients With Arrhythmia

Technique	Comment
<ul style="list-style-type: none">• Heart rate and/or rhythm control before scanning	<ul style="list-style-type: none">• Use beta-blockers or other antiarrhythmic medication
<ul style="list-style-type: none">• Use Arrhythmia Rejection	<ul style="list-style-type: none">• Increases breath-hold time
<ul style="list-style-type: none">• Use Prospective triggering	<ul style="list-style-type: none">• Reduces SNR
<ul style="list-style-type: none">• Use Real-time imaging	<ul style="list-style-type: none">• Reduces temporal and spatial resolution as well as SNR

Function Module

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 $\leq 45\text{ms}$**

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 $\leq 45\text{ms}$**

Function Module

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.

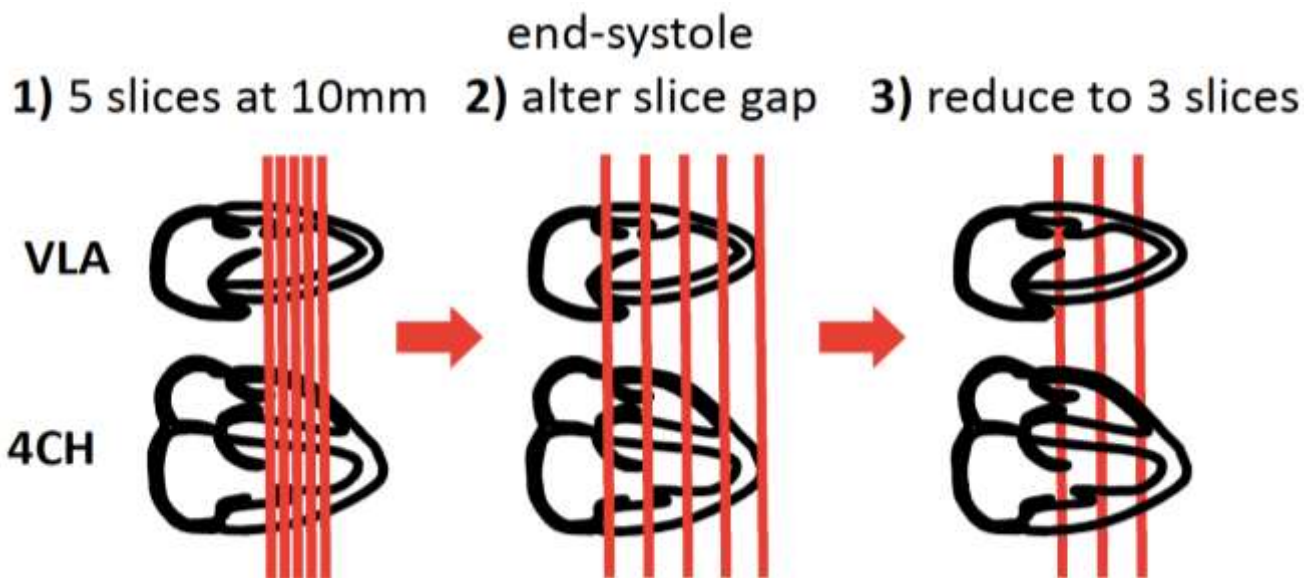
Perfusion Module

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**

Perfusion Module

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**



Early and Late Gd

Enhancement Module

1/2

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

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Early and Late Gd

Enhancement Module

2/2

Tips & Tricks

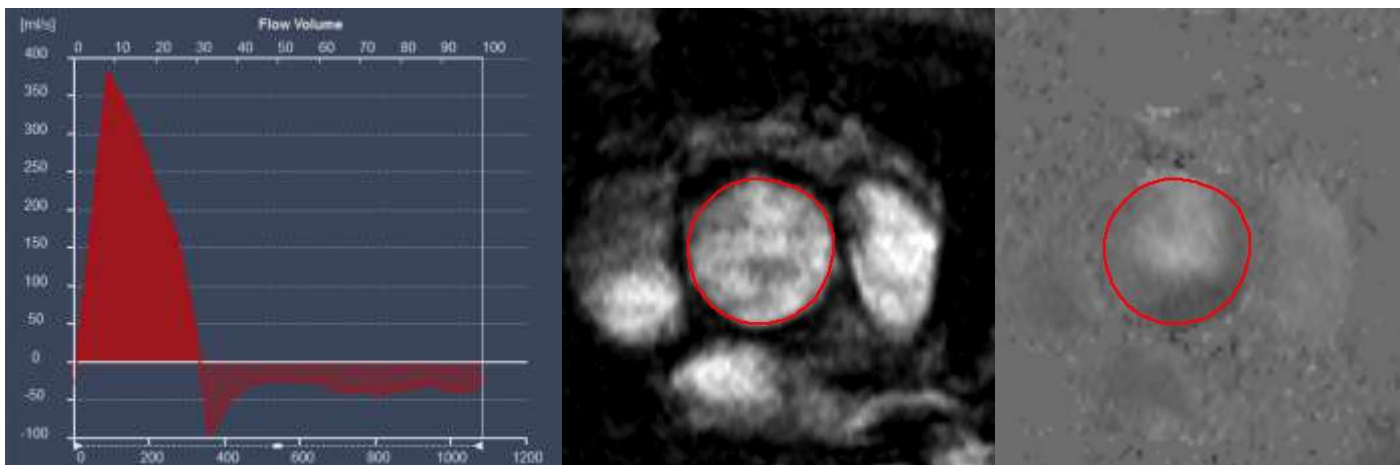
1. **Scan in mid- or late-diastole** to minimize motion artefacts
2. Use **saturation bands** across the spinal column and the anterior chest wall to reduce ghosting artefacts
3. **Late enhancement on images:**
 - Use **“Phase Swap”** (changing the phase encoding direction) to confirm pathology/detect 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

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Phase Contrast Velocity Encoded Module

1/2

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: 150cm/s**
 - **Normal right-sided 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**



Volume time curve from flow velocity encoding through the ascending aorta in a patient with severe aortic regurgitation

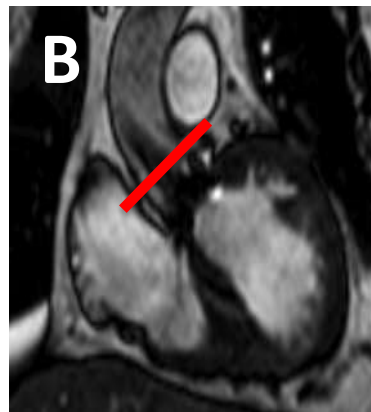
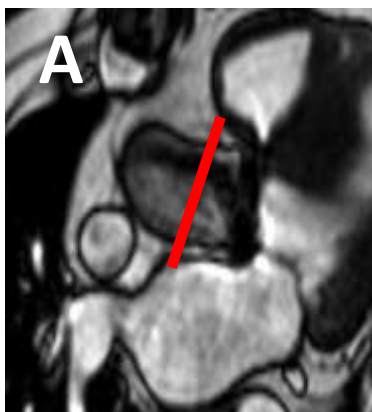
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Phase Contrast Velocity Encoded Module

2/2

Tips & Tricks

- 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
- Avoid underestimation of velocities. Check:**
 - Adequate temporal resolution (phases)
 - Free-breathing acquisition: 30 phases
 - Breath-hold acquisition: 20-25 phases
- Rotate FOV - orthogonal to the direction of flow
- Slice thickness: <7mm

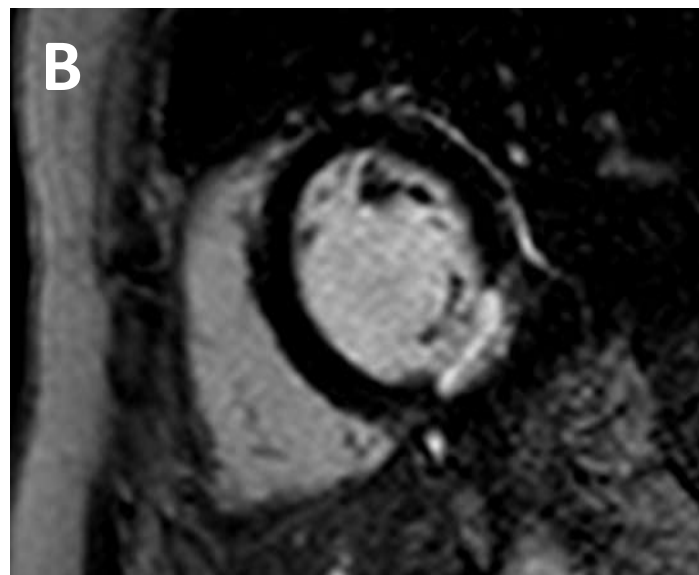
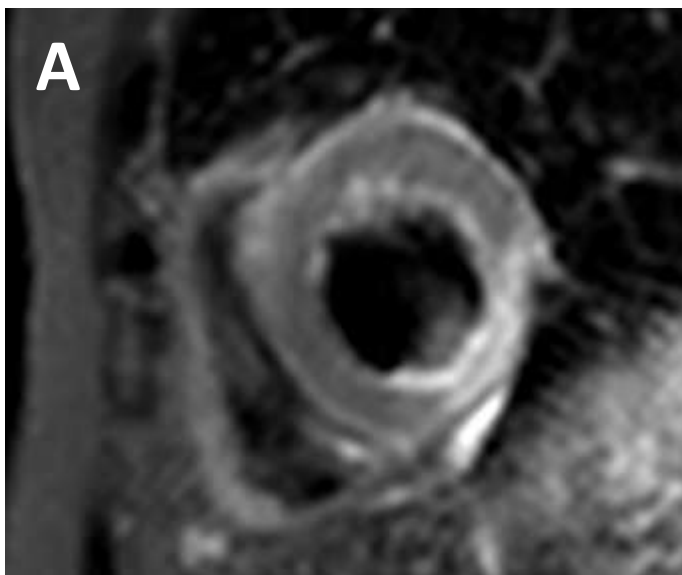


Sagittal (A) and coronal (B) slice positioning for aortic stenosis

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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 coil-related signal differences
6. Slow flow artefacts may cause high signal at endocardial border



Myocardial infarction with inferior edema on T2w images (A) and LGE (B)

Angiography Module

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

Coronary Artery Imaging Module

1/2

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

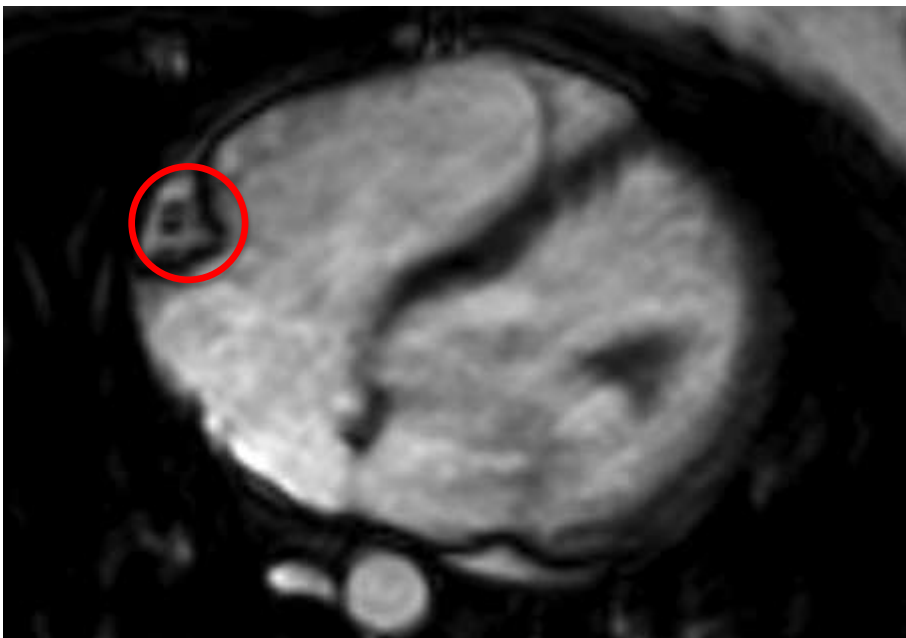
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Coronary Artery Imaging Module

2/2

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** (≥ 90 bpm): 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

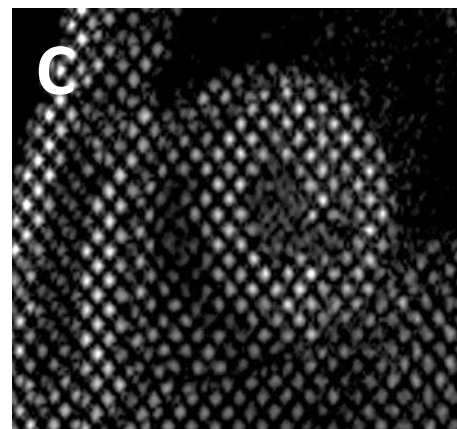
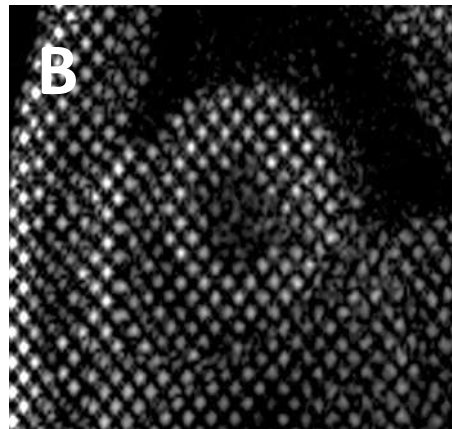
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Tagging Module

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



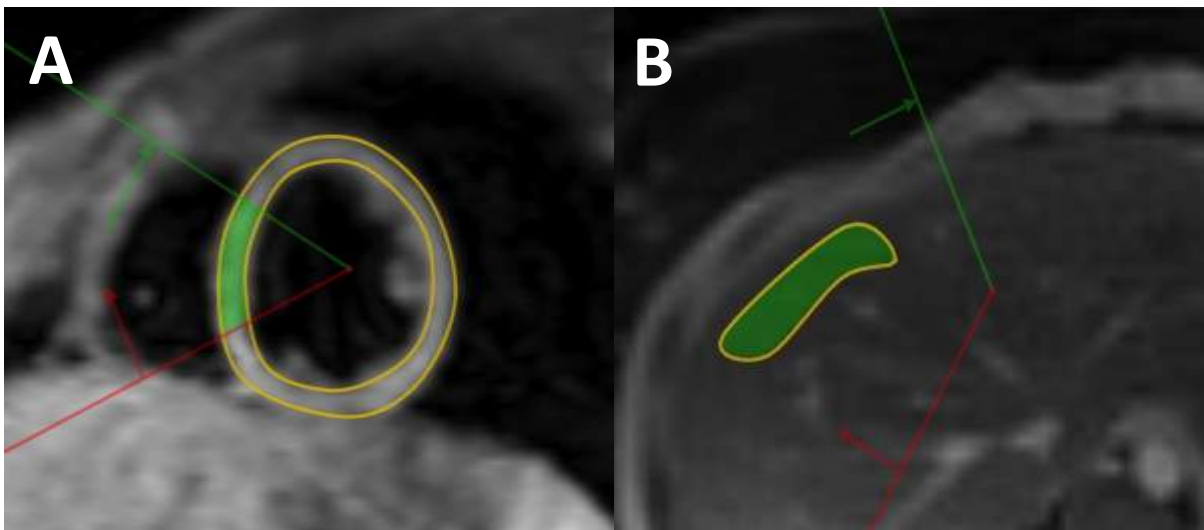
Apical (A), mid-ventricular (B) and basal (C) grid-tagging

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

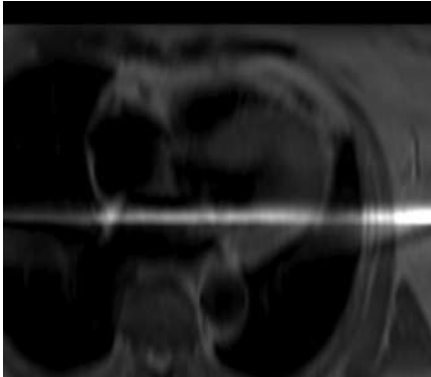
1. **Ensure good patient breath-holding for the heart and the liver scans** by coaching as the scan duration is long
2. **Make sure the septum is of good image quality** as this is where quantification is most reproducible
3. Position the transverse liver slice correctly:
 - Avoid large hepatic vessels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



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

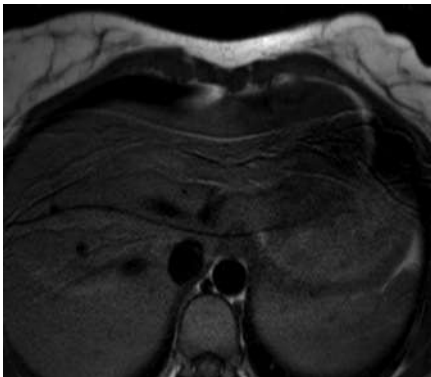
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Artefacts



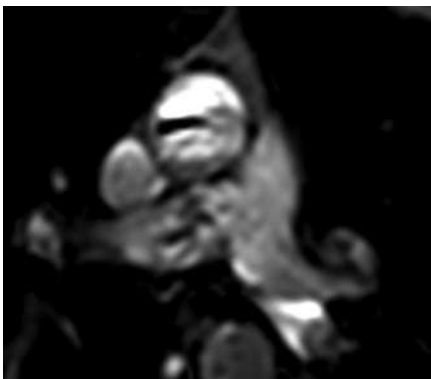
Wrapping artefact (fold-over, back-folding)

- 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)

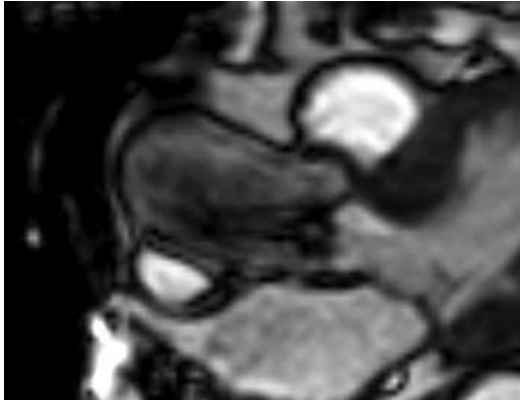
- Strict breath-holding plus acceleration techniques
- Respiratory gating or navigator echoes
- Swap phase and frequency direction
- Use selective tissue saturation bands to suppress the signal from the anterior abdominal wall



Ghosting artefact from motion (pulsatile flow)

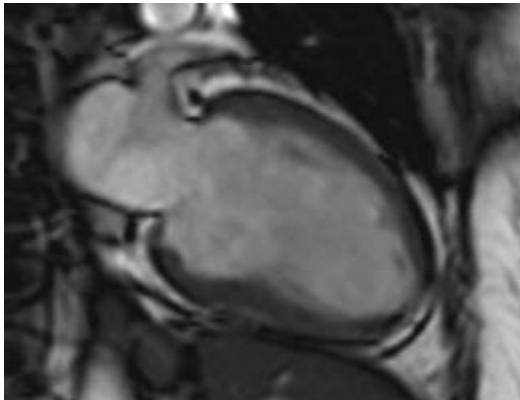
- Use ECG triggering / gating
- Use flow compensation (gradient moment nulling, gradient motion rephasing)
- Use selective tissue saturation bands to suppress the blood signal
- Swap phase and frequency direction

Artefacts



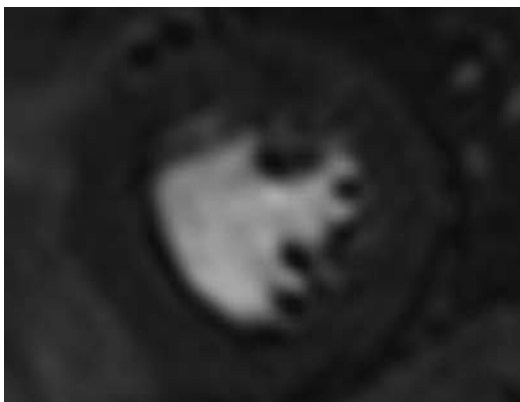
Flow-related signal loss and flow jets

- Reduce echo time
- Use flow compensation
- Use bSSFP acquisition



Chemical shift artefact

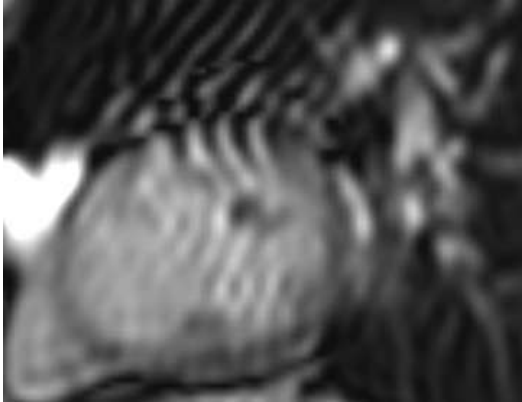
- Compare with other images as they are sequence dependent



Dark rim artefact

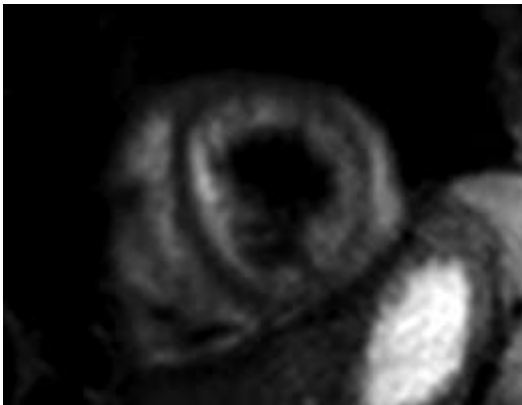
- Often seen in perfusion imaging
- Reduce contrast dose/infusion speed
- Increase in-plane spatial resolution

Artefacts



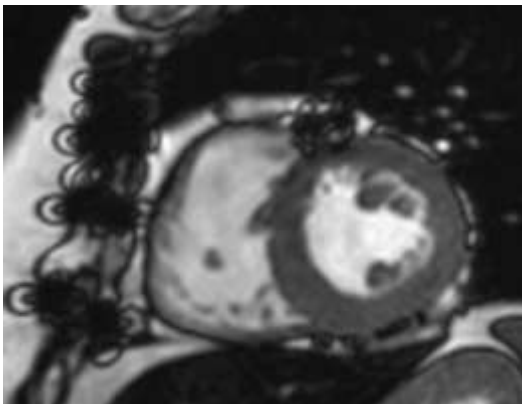
Radiofrequency interference artefact

- 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

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**)

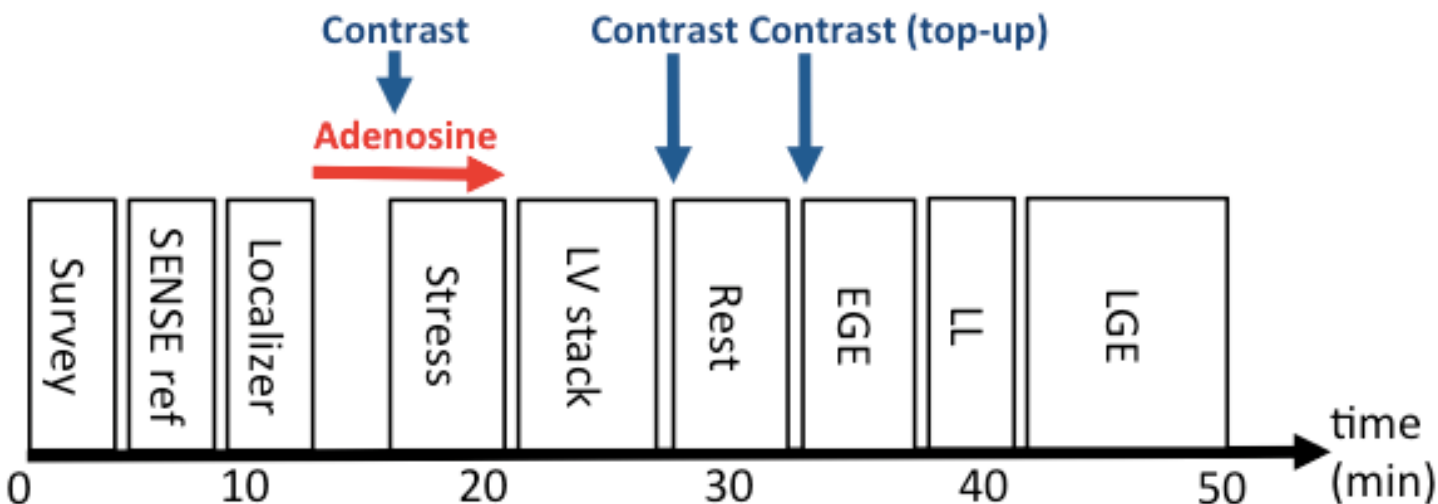
Perfusion

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



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 = $0.85 \times (220 - \text{age})$
 - 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

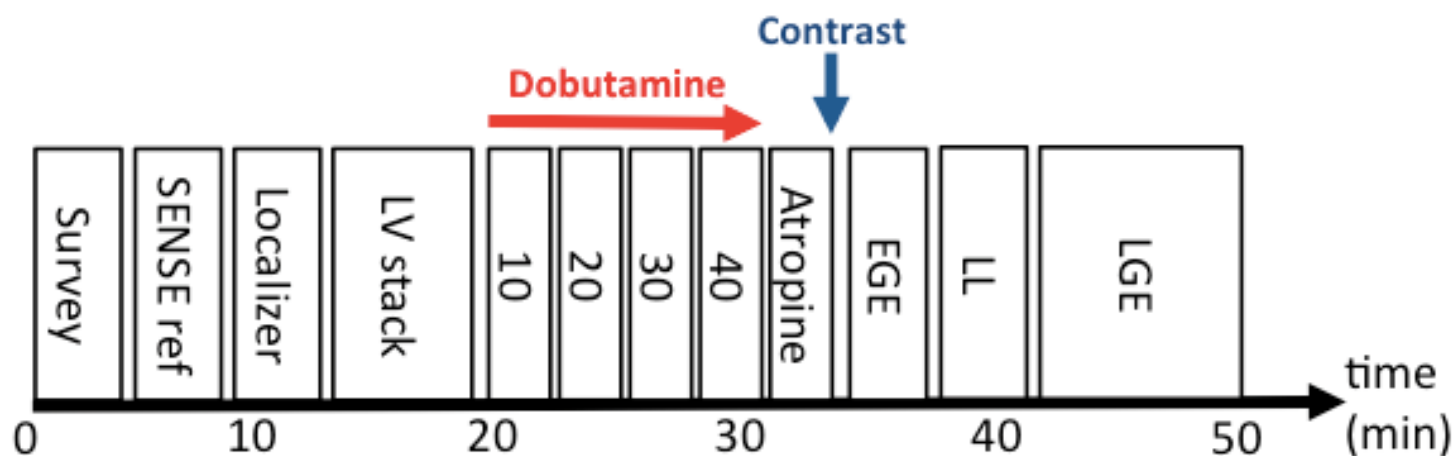
Wall Motion

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



Wall Motion

Ischemic Conditions	Myocardial Wall Motion		
	Rest	Low-Dose Dobutamine	High-Dose Dobutamine
Normal			
Ischemia			
Hibernation			
Subendocardial Scar			
Transmural scar			

Wall Motion Score Index (WMSI)

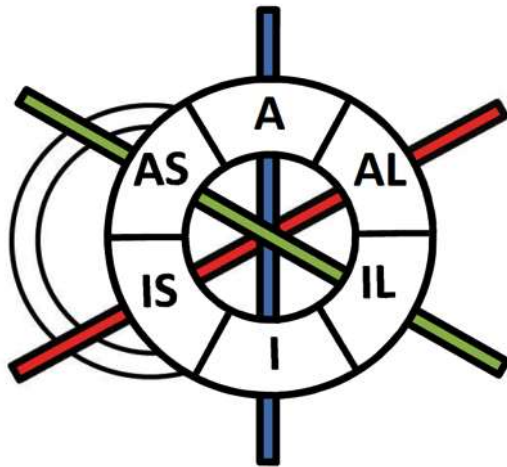
(sum of wall motion scores / number of segments)

Normal	1
Hypokinetic	2
Akinetic	3
Dyskinetic	4
Aneurysmal	5

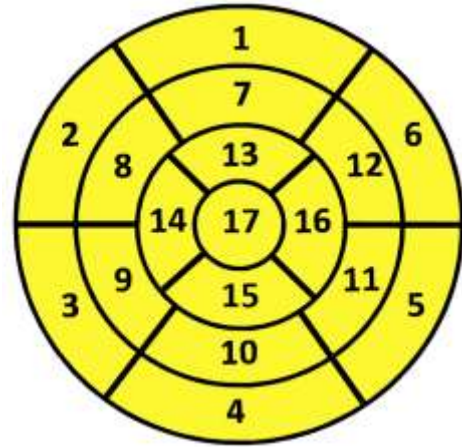
A WMSI of 1 is considered normal



17 Segment Model

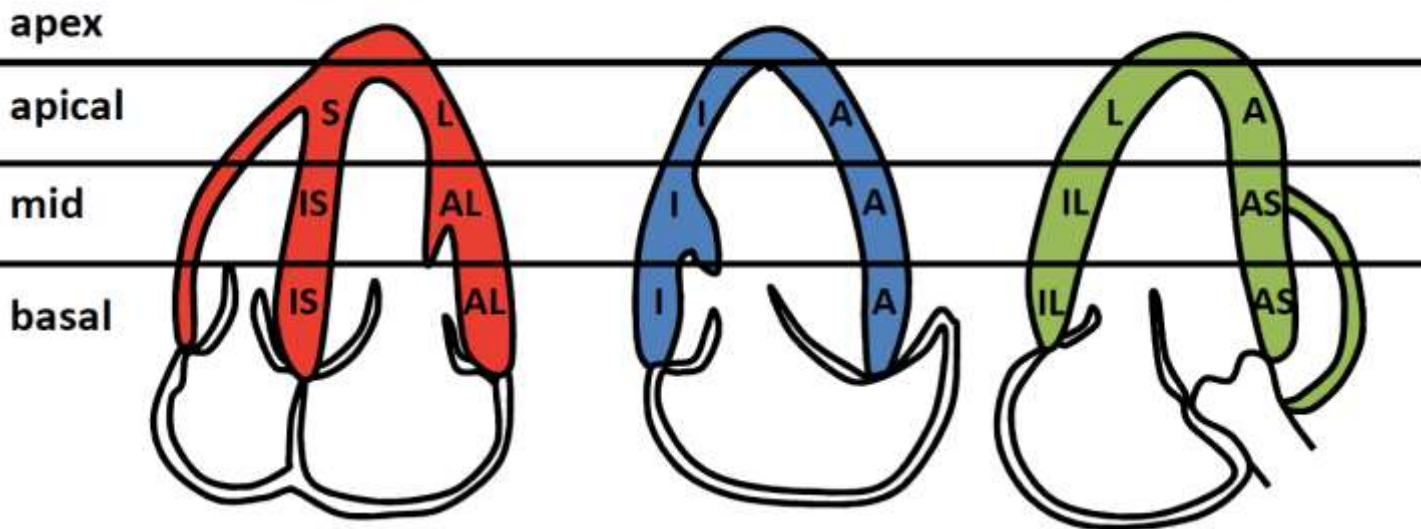


4 chamber



2 chamber

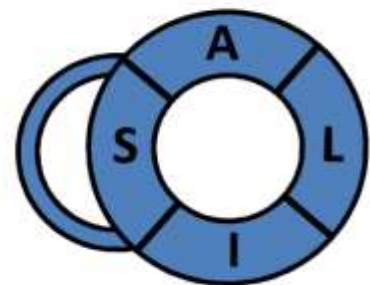
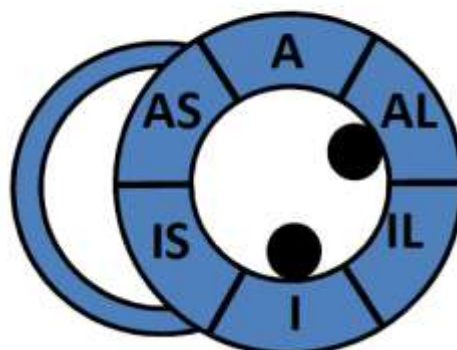
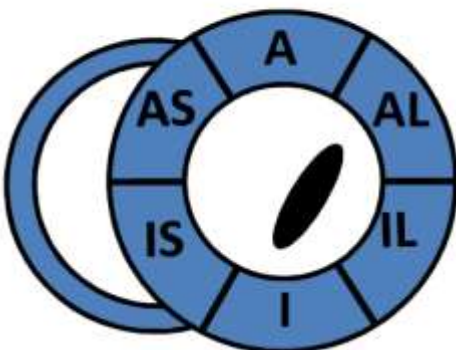
3 chamber



basal

mid

apical



Modified from reference 4)

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-anterseptal AS; 9: mid-inferoseptal IS; 10: mid-inferior I; 11: mid-inferolateral IL; 12: mid-antrolateral AL; 13: apical anterior A; 14: apical septal S; 15: apical inferior I; 16: apical lateral L; 17: apex

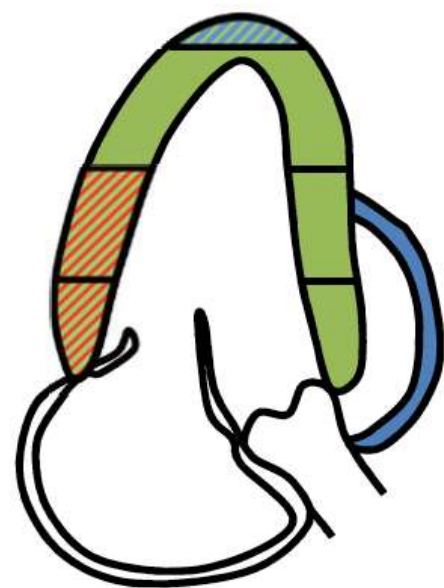
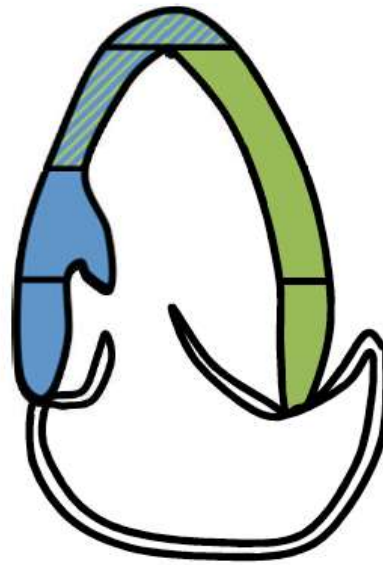
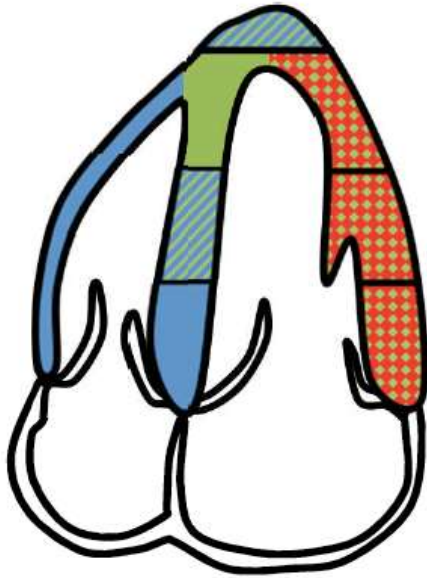
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Coronary Supply

4 chamber

2 chamber

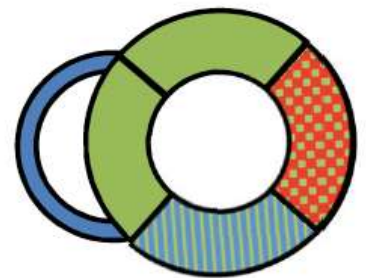
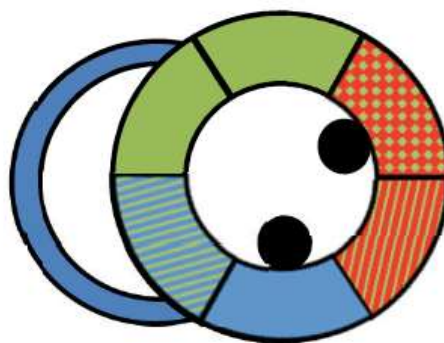
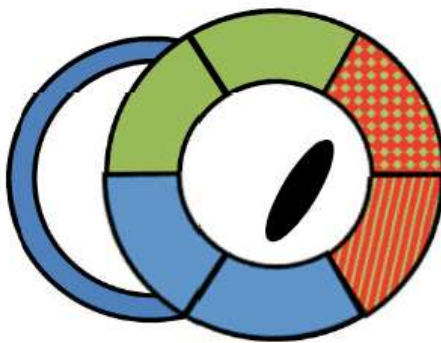
3 chamber



basal

mid

apical



LAD



LAD or CX



CX



LAD or RCA



RCA



CX or RCA

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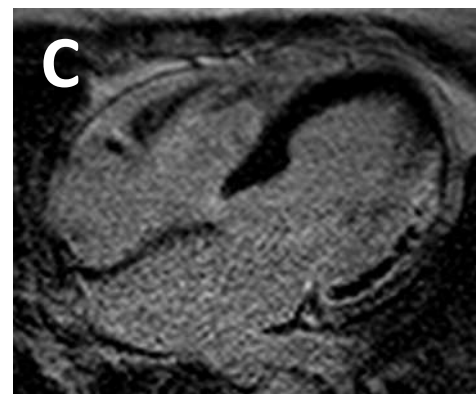
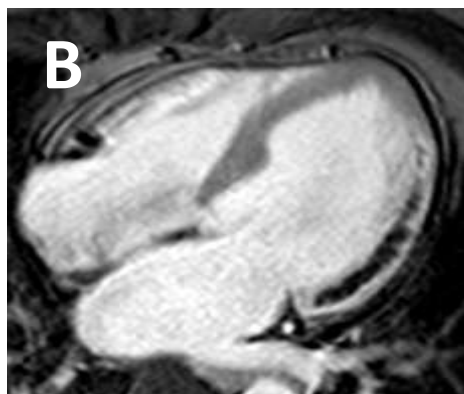
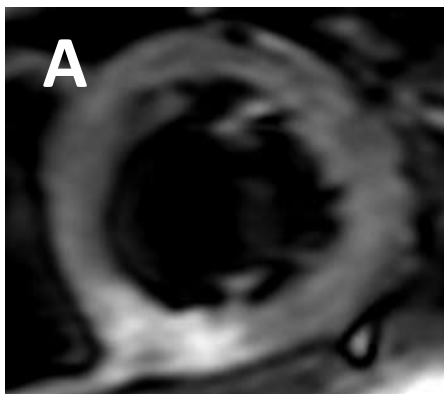
Acute Myocardial Infarction

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

Acute Myocardial Infarction

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

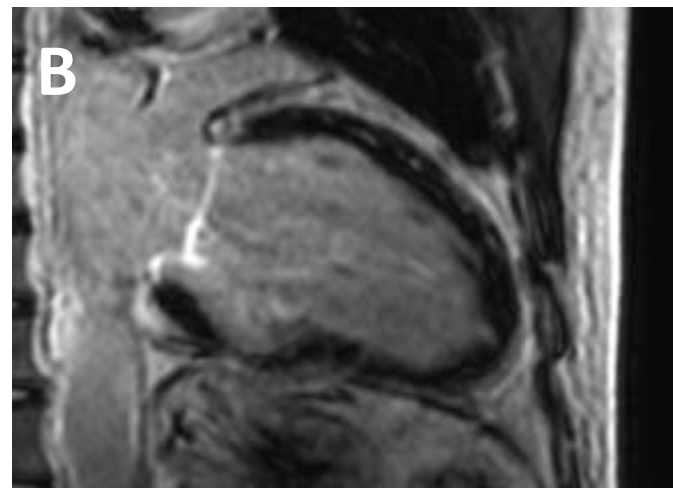
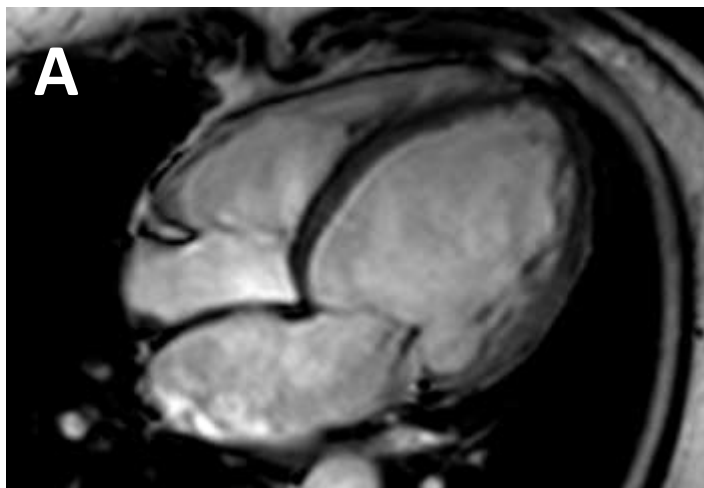
Dilated Cardiomyopathy^{1/2}

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**



Dilated LV with MR (A) and mid-wall fibrosis on LGE images (B)

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Dilated Cardiomyopathy ^{2/2}

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

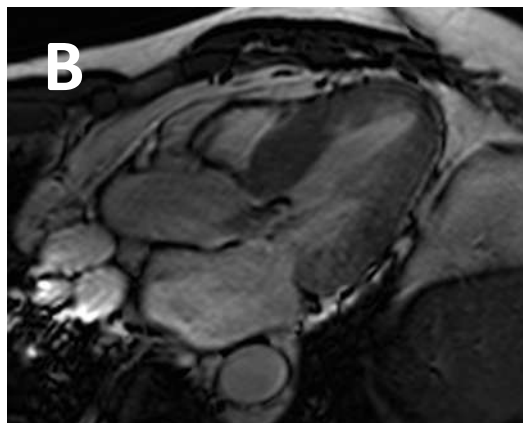
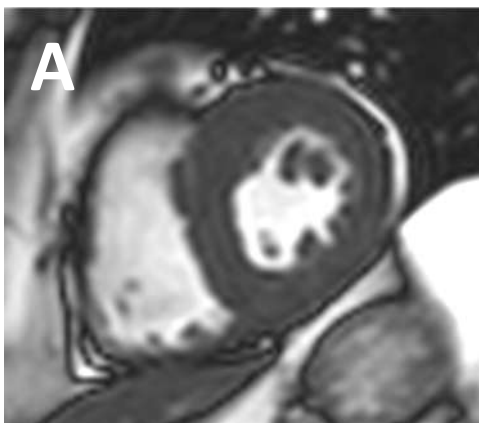
1/2

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)

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Hypertrophic Cardiomyopathy

2/2

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 $\geq 30\text{mm}$
 - 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

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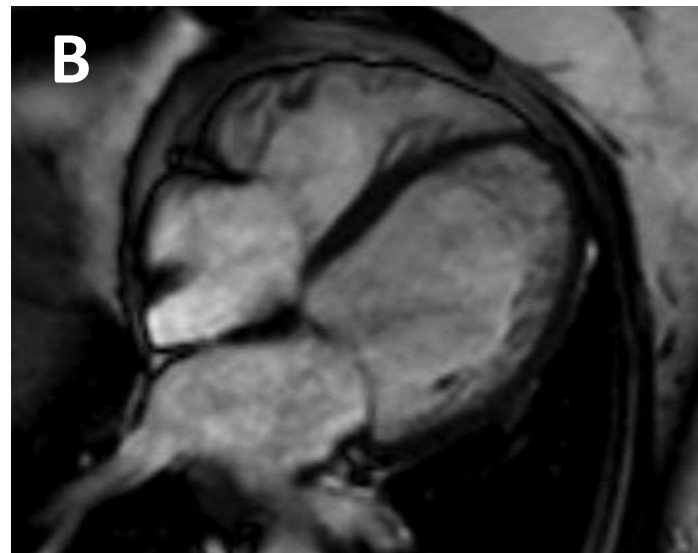
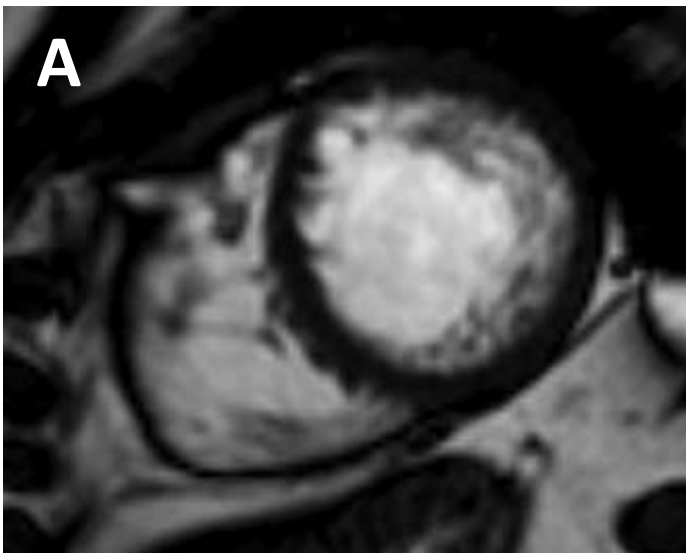
Left Ventricular Non- 1/2 Compaction Cardiomyopathy

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**



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

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Left Ventricular Non-2/2 Compaction Cardiomyopathy

Key Points

- 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
- LGE** may represent **severe or late forms** of LVNC
- 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

Ventricular Cardiomyopathy

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**

Arrhythmogenic Right Ventricular Cardiomyopathy 2/5

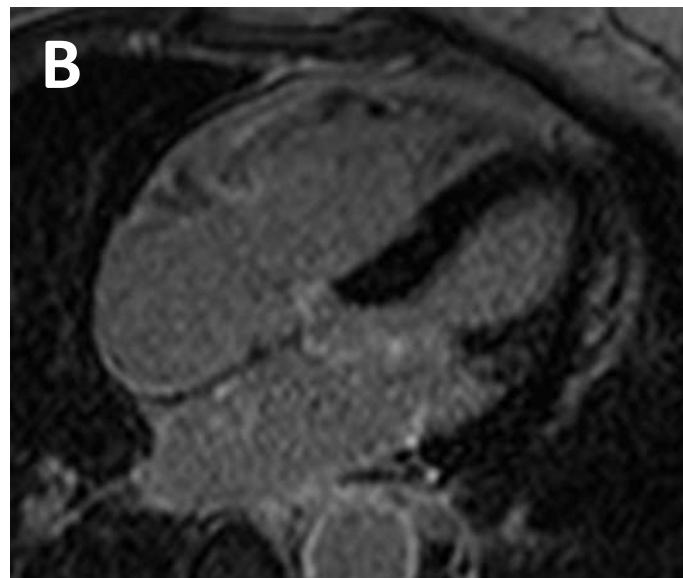
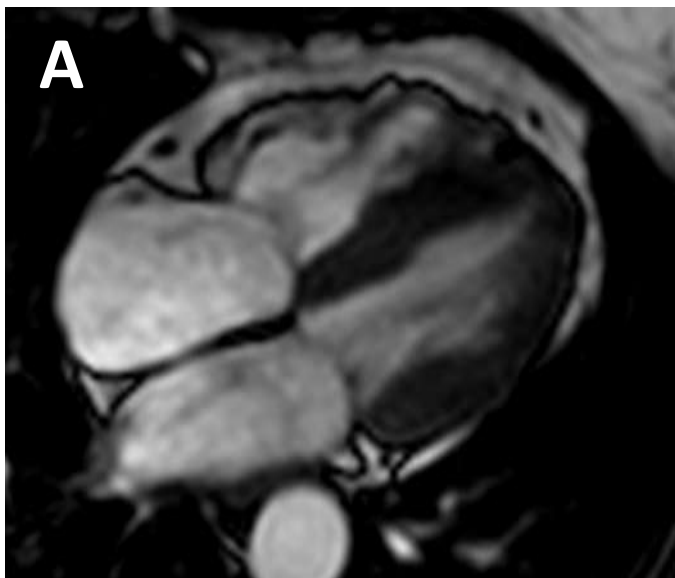
Ventricular Cardiomyopathy

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)

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Diagnostic Criteria

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 EDV 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 EDV to BSA ≥ 100 to $< 110 \text{ mL/m}^2$ (male) or ≥ 90 to $< 100 \text{ mL/m}^2$ (female)
 - or RV EF $> 40\%$ 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 ≥ 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

Diagnostic Criteria

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 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 \geq 1 of 3 parameters in the absence of a QRS duration of \geq 110ms on the standard ECG
 - Filtered QRS duration (fQRS) \geq 114ms
 - Duration of terminal QRS $<$ 40 μ V (low-amplitude signal duration) \geq 38ms
 - Root-mean-square voltage of terminal 40ms \leq 20 μ V
 - Terminal activation duration of QRS \geq 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

Diagnostic Criteria

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

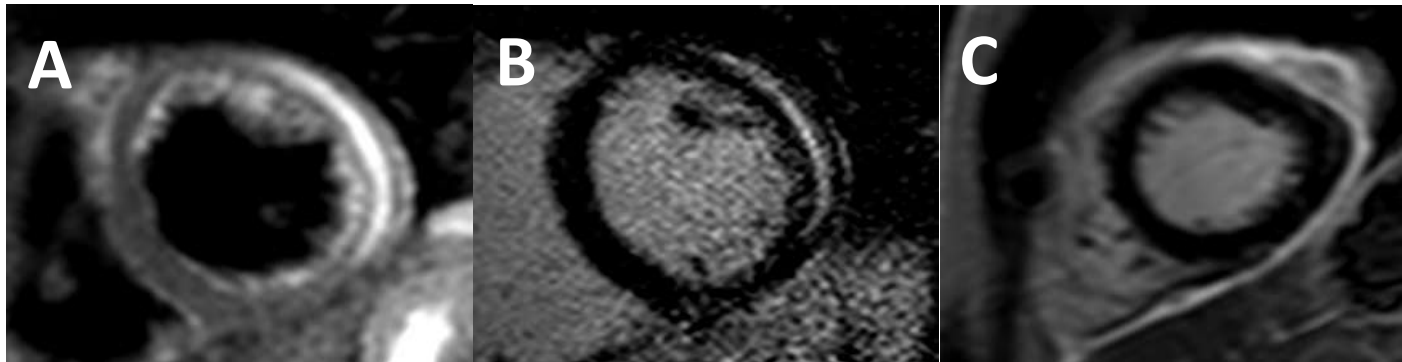
Myocarditis / Pericarditis ^{1/2}

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).

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Myocarditis / Pericarditis ^{2/2}

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 $\geq 45\%$
- **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)

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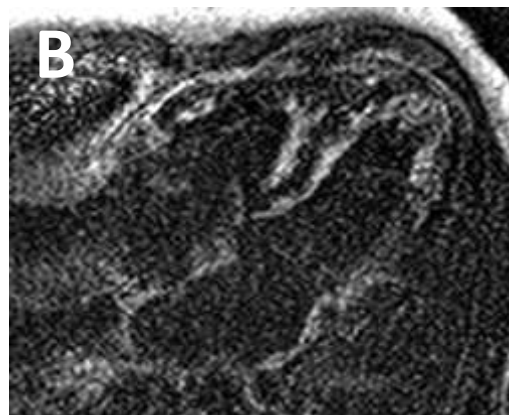
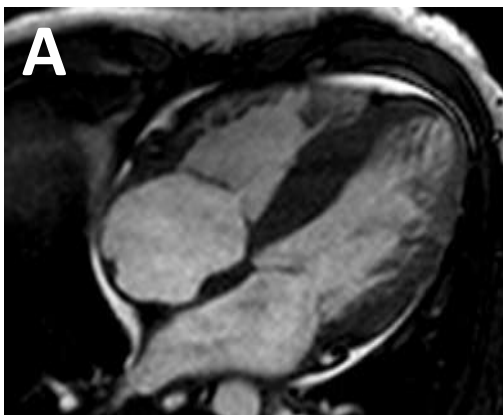
Amyloidosis

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)

Amyloidosis

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

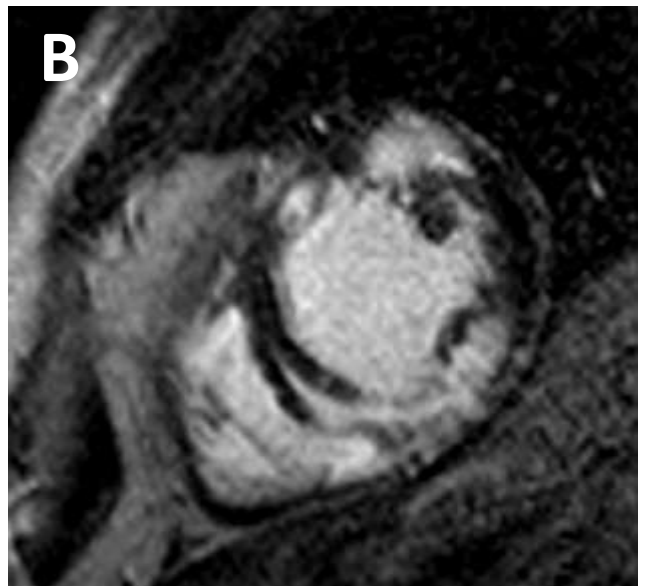
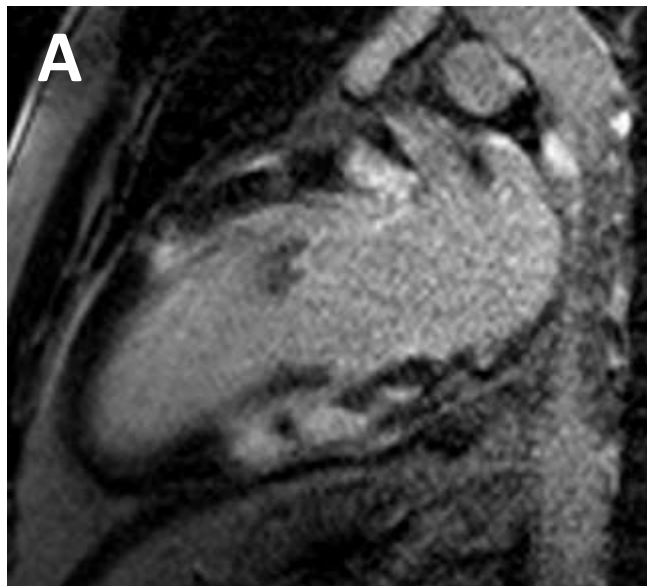
Sarcoidosis

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**



Sarcoidosis

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

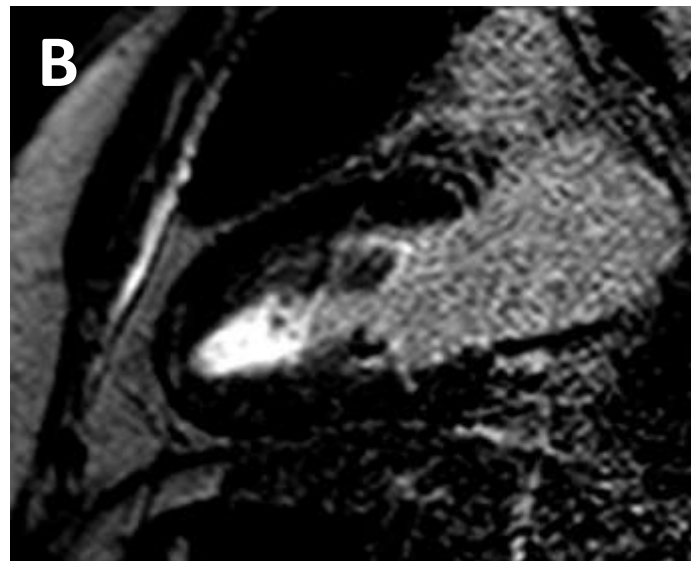
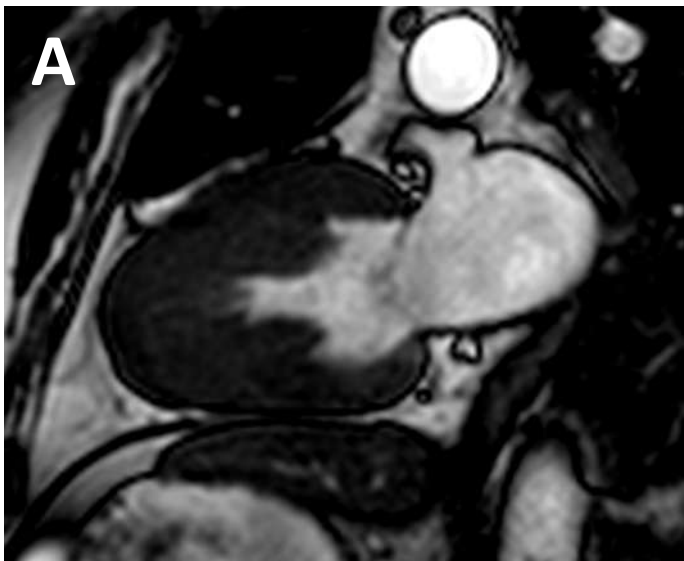
Endomyocardial Fibrosis^{1/2}

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**



Hypertrophied LV (A) with endocardial fibrosis on LGE (B)

Endomyocardial Fibrosis

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)

Tako-Tsubo Cardiomyopathy

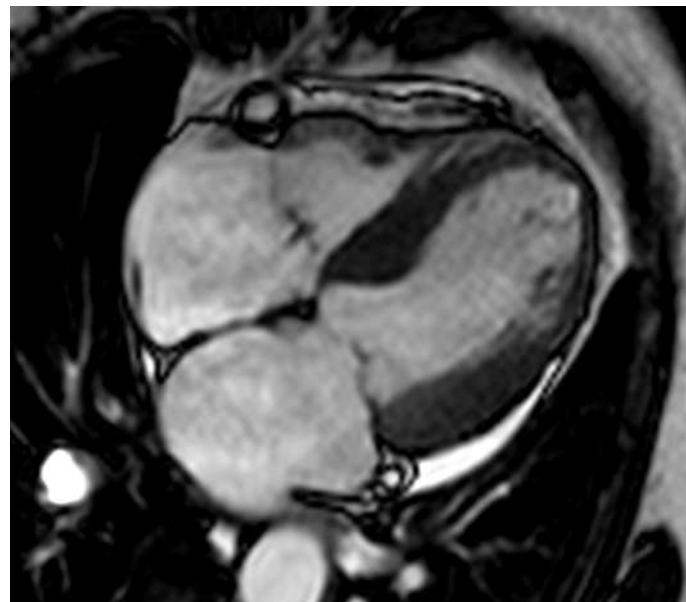
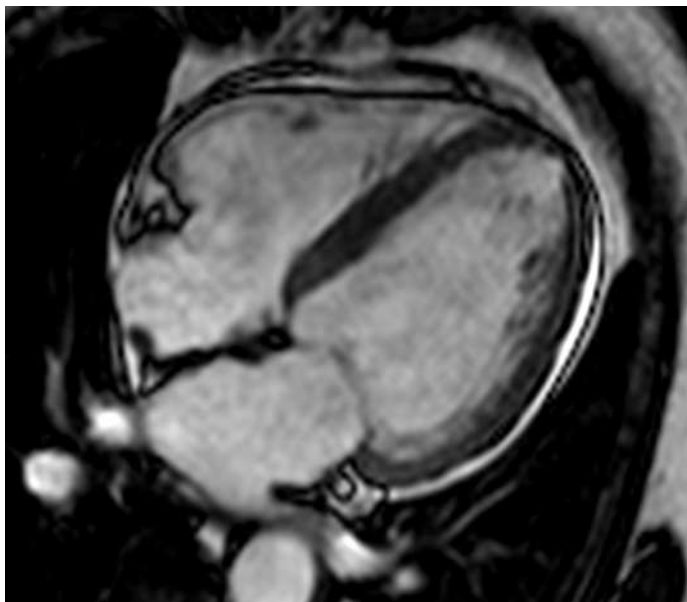
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Protocol

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

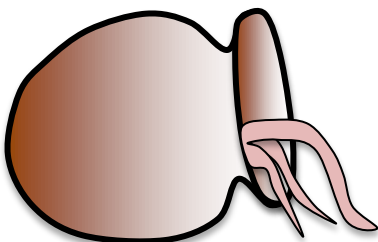
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Tako-Tsubo Cardiomyopathy

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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



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Pericardial Effusion

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**
 - 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

Pericardial Effusion

Key Points

- Pericardial tamponade is a clinical diagnosis**
 - Even a small and focal effusion can be haemodynamically significant
- Signs of tamponade:**
 - RA / LA collapse, RV / LV collapse
 - Septal shift towards LV during inspiration
- Typical causes of pericardial effusion:**
 - Global: uremic, infectious, myxedema, 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	↑

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 $\geq 3\text{mm}$**
4. Presence or absence of **RV diastolic collapse**
5. **LGE** enhancement in RV, LV and pericardium

Constrictive Pericarditis

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

Anomalous Coronary Arteries

1/2

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

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Anomalous Coronary Arteries

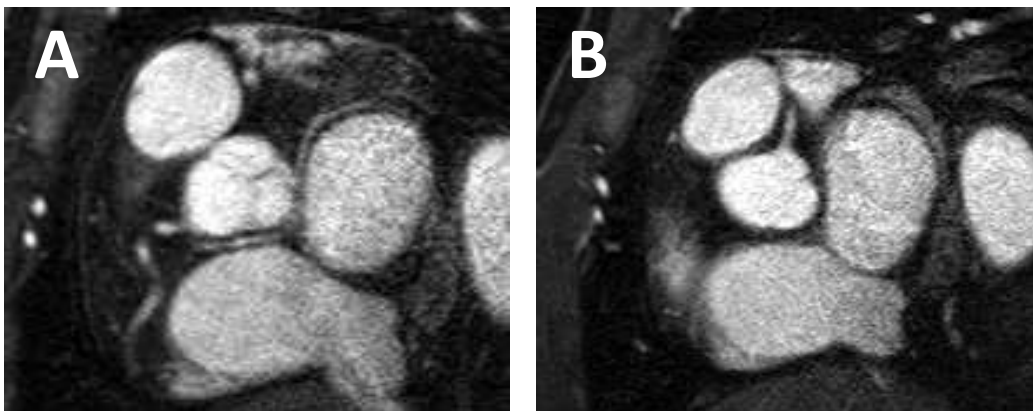
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Key Points

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-aortic course (A). Normal origin of the RCA (B)

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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**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
2. **Aorta position** (left or right) and **tortuosity**
3. **Atherosclerosis, aneurysm, dissection, inflammation**
4. **Aortic flow**
5. **Associated aortic valvular stenosis or regurgitation**

Aortic Disease

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

Valvular Heart Disease

Key Points

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)

Pulse sequence

Indication

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

Valvular Heart Disease

Calculation of regurgitant volume in SINGLE valve disease

Aortic regurgitation	<ul style="list-style-type: none"> • Regurgitation volume/fraction from phase contrast VENC above aortic valve • Alternatively $LV\ SV - RV\ SV$
Mitral regurgitation	<ul style="list-style-type: none"> • SV from phase contrast VENC above aortic valve – LV SV • Alternatively $LV\ SV - RV\ SV$
Pulmonary regurgitation	<ul style="list-style-type: none"> • Regurgitation volume/fraction from phase contrast VENC above pulmonary valve • $RV\ SV - LV\ SV$
Tricuspid regurgitation	<ul style="list-style-type: none"> • 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

Valvular Heart Disease

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, pediculated
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

Cardiac Mass	T1w*	T2w*	LGE
Pseudotumors			
Thrombus	Low (high if recent)	Low (high if recent)	No uptake†
Pericardial cyst	Low	High	No uptake
Benign			
Myxoma	Isointense	High	Heterogeneous
Lipoma	High‡	High‡	No uptake
Fibroma	Isointense	Low	Hyperenhanced
Rhabdomyoma	Isointense	Isointense/high	No/min. uptake
Malignant			
Angiosarcoma	Heterogenous	Heterogenous	Heterogeneous
Rhabdo- myosarcoma	Isointense	Hyperintense	Homogeneous
Undifferentiated sarcoma	Isointense	Hyperintense	Heterogeneous/V ariable
Lymphoma	Isointense	Isointense	No/min. uptake
Metastasis §	Low	High	Heterogeneous

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.

Common MR Terminology

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

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Abbreviations

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

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