

Effusion and valves

From CMR Pocket Guide First Edition 2013

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

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

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

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

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

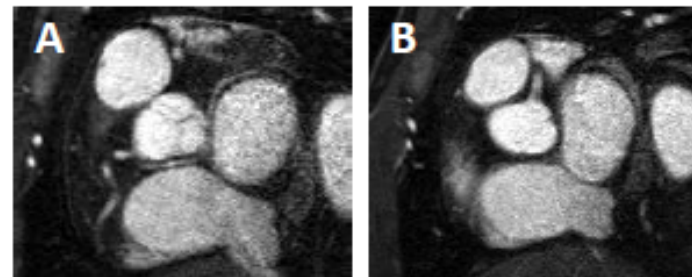
5. Anomalous collateral vessels

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)

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**

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

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

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)**

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 variable
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.