Cardiovascular Magnetic Resonance
ESC Working Group

EUROPEAN SOCIETY OF CARDIOLOGY®

EuroCMR exam syllabus

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The EuroCMR exam syllabus will be reviewed and amended on a regular basis. The EuroCMR exam syllabus provides detailed information about what the candidate is expected to be familiar with and can thus guide in the preparation for the exam. The EuroCMR exam syllabus is not exhaustive and the candidate should be aware that questions may be asked in the EuroCMR exam that does not map to the current version of the syllabus.

1. Physics, safety, devices, CMR methodology and anatomy
   - 1.1. Physics
     - 1.1.1. Basic magnetic resonance physics
       - 1.1.1.1. Magnetic properties of hydrogen nuclei: Nuclear spin, Precession, Resonance, Larmor frequency, Excitation
       - 1.1.1.2. Relaxation mechanisms: spin-lattice relaxation and spin-spin relaxation (T1 and T2 relaxation times)
       - 1.1.1.3. Tissue properties: T1, T2 and T2*
       - 1.1.1.4. Different field strengths (1.5T and 3T, earth ~0.5 G, 1T=10kG)
     - 1.1.2. Signal spatial encoding
       - 1.1.2.1. Types of main static magnets (Principles of superconducting magnets, possible field strengths, extent of fringe field, field homogeneity, open and closed magnets)
       - 1.1.2.2. Scanner parameters: B_0 field strength, gradient strength, rise time
       - 1.1.2.3. Radiofrequency system: Transmit and receive coils, phased array coils, surface coils, RF pulses, need for Faraday cage
       - 1.1.2.4. Magnetic field gradients: relationship between spatial encoding and spatial frequency
       - 1.1.2.5. 2D signal encoding: Slice selection, Phase and frequency encodings
       - 1.1.2.6. Interpreting spatial encoding in MRI: k-space properties and Fourier transform
       - 1.1.2.7. Relationships between contrast, spatial resolution, matrix size, acquisition time, field of view, receiver bandwidth and k-space
       - 1.1.2.8. 3D spatial encoding
     - 1.1.3. Basic pulse sequences
       - 1.1.3.1. Basic excitation pulses: 90° pulse, 180° refocusing and inversion pulses (selective vs non-selective)
       - 1.1.3.2. Basic gradient and spin echoes
       - 1.1.3.3. Signal-to-noise ratio (SNR) and parameters affecting it (e.g. voxel size, signal averages, receiver BW, B_0,partial k-space acquisition, 2-D vs 3-D)
       - 1.1.3.4. MRI tissue contrasts: Relation between TR, TE, flip angle and T1- weighting, T2- weighting, proton density-weighting imaging
       - 1.1.3.5. Prepulses and their effect on tissue contrast: Inversion Recovery, STIR, fat suppression, T2-preparation

- 1.1.3.6. Basic pulse sequence diagrams
- 1.1.4. Advanced and fast pulse sequences
  - 1.1.4.1. Fast spin echo sequences
  - 1.1.4.2. Spoiled gradient echo, balanced steady state free precession (SSFP)
  - 1.1.4.3. Echo planar imaging (EPI)
  - 1.1.4.4. Hybrid sequences (spin echo and gradient echo)
  - 1.1.4.5. Spiral Imaging
  - 1.1.4.6. Sequence acronyms: generic sequence names and vendor specific names
- 1.1.5. Parallel imaging
  - 1.1.5.1. Radiofrequency system for parallel imaging: Phased array coils
  - 1.1.5.2. Benefits and drawbacks of parallel imaging: scan time, SNR, noise level
  - 1.1.5.3. Image acquisition and reconstruction methods: image domain, frequency domain, spatio-temporal domain
  - 1.1.5.4. Main applications
- 1.1.6. MR Angiography flow imaging
  - 1.1.6.1. Flow phenomena in MRI
  - 1.1.6.2. Flow compensation
  - 1.1.6.3. Non-contrast magnetic resonance angiography methods: Time-of-flight MR angiography, Phase-contrast MRA
  - 1.1.6.4. Phase contrast velocity imaging
  - 1.1.6.5. Contrast-enhanced MRA techniques using contrast agents
- 1.1.7. Cardiac MRI applications
  - 1.1.7.1. Cardiac triggering and sequence synchronization, segmented acquisitions
  - 1.1.7.2. Respiratory compensation: breath-holding, respiratory navigators, respiratory gating
  - 1.1.7.3. Bright blood and dark blood sequences
  - 1.1.7.4. Sequences for cardiac morphology, function, tagging and perfusion imaging: type of sequences, optimisation of image quality and scan times
  - 1.1.7.5. Sequences for myocardial delayed enhancement MRI: sequence and image quality optimization
  - 1.1.7.6. Coronary/Bypass graft MRA, Phase contrast velocity mapping
- 1.1.8. Image quality and artefacts
  - 1.1.8.1. Factors influencing the signal-to-noise ratio and their interdependence
  - 1.1.8.2. Trade-off between scan time, signal-to-noise ration and spatial resolution
  - 1.1.8.3. MRI artefacts, their sources, effects on the image quality and how to reduce them
  - 1.1.8.4. Physiological motion: origin of artefacts and remedies (ghosting)
  - 1.1.8.5. Imaging of sedated patients and patients with arrhythmias
  - 1.1.8.6. Parallel imaging: foldover, aliasing, noise amplification
  - 1.1.8.7. Magnetic susceptibility and metal artefacts
  - 1.1.8.8. Fat-water chemical shift
  - 1.1.8.9. Truncation, Gibbs artefacts
  - 1.1.8.10. Equipment artefacts
  - 1.1.8.11. Shimming
- 1.2. Safety/setup/device
  - 1.2.1.1. MRI setup
  - 1.2.1.2. Main magnet
  - 1.2.1.3. Radio frequency transmit/receive systems
  - 1.2.1.4. Magnetic field gradients systems
  - 1.2.1.5. Computer systems
- 1.2.2. MRI safety

- 1.2.2.1. Static field biological effects
- 1.2.2.2. Safety zones for MRI facility, I-IV (USA)
- 1.2.2.3. Radiofrequency field biological effects
  - 1.2.2.3.1. Sequence-related patient heating
  - 1.2.2.3.2. Factors affecting SAR and how to reduce it
- 1.2.2.4. Gradient fields biological effects
  - 1.2.2.4.1. Acoustic noise during MRI scanning
  - 1.2.2.4.2. Peripheral nerve stimulation
- 1.2.2.5. Precautions prior and during an MRI examination
- 1.2.2.6. Cryogen safety
- 1.2.2.7. Pregnancy
- 1.2.2.8. Emergency procedures
- 1.2.2.9. Contrast agents: families of contrast agents, effect on relaxation times, contraindications (renal failure-NSF, allergy, pregnancy) and main applications

1.2.3.MRI device

- 1.2.3.1. MRI device safety classifications
- 1.2.3.2. Passive implants and devices: valve repairs, stents, coils, aneurysm clips,…
- 1.2.3.3. Active devices: insertable loop recorders, ICD, pacemakers, abandoned leads
  - 1.2.3.3.1. Safe, conditional and unsafe pacemakers
  - 1.2.3.3.2. Precautions post and post scans
- 1.2.3.4. Safety at 1.5T and 3T

1.3. CMR Methodology

1.3.1.Cardiac anatomy including the AHA/ACC 17 segment model nomenclature

1.3.2.Cardiac function

- 1.3.2.1. LV volumes
- 1.3.2.2. RV volumes
- 1.3.2.3. Regional wall motion abnormalities (tagging, DENSE, tissue phase mapping etc)

1.3.3.Tissue characterisation

- 1.3.3.1. Non-contrast techniques
  - 1.3.3.1.1. T2-STIR
- 1.3.3.2. Contrast-enhanced techniques
  - 1.3.3.2.1. Early gadolinium enhancement
  - 1.3.3.2.2. Late gadolinium enhancement

1.3.4.CMR stress imaging

- 1.3.4.1. Myocardial perfusion imaging
- 1.3.4.2. Dobutamine stress CMR

1.3.5.Blood flow

2. Ischaemic heart disease

2.1. Chronic ischemic heart disease

2.1.1.Chronic myocardial infarction

- 2.1.1.1. Infarct imaging with LGE
- 2.1.1.2. Infarct imaging with T1 mapping
- 2.1.1.3. RV infarction
- 2.1.1.4. Assessment of ventricular remodelling
  - 2.1.1.4.1. Left ventricle
  - 2.1.1.4.2. Right ventricle

2.1.2.Viability assessment

- 2.1.2.1. Late gadolinium enhancement
  - 2.1.2.1.1. Sequences & scanning technique
  - 2.1.2.1.2. Clinical validation
  - 2.1.2.1.3. Quantification of scarred myocardium/analysis methods
  - 2.1.2.1.4. Clinical application (prediction of regional or global functional recovery)
- 2.1.2.2. Differentiation from causes of non-ischemic scarring

- 2.1.2.3. Low-dose dobutamine stress
  - 2.1.2.3.1. Protocol/ scanning technique
  - 2.1.2.3.2. Clinical validation & application (definition of 'viable' response)
- 2.1.3.Ischemia testing
  - 2.1.3.1. Stress agents
    - 2.1.3.1.1. Dobutamine
    - 2.1.3.1.2. Adenosine
    - 2.1.3.1.3. Regadenoson
    - 2.1.3.1.4. Dipyridamole
  - 2.1.3.2. First pass perfusion imaging during stress & rest using a contrast agent
    - 2.1.3.2.1. Sequences & scanning technique
    - 2.1.3.2.2. Indications
    - 2.1.3.2.3. Contraindications
    - 2.1.3.2.4. Clinical validation& application
    - 2.1.3.2.5. Artefacts & pitfalls
    - 2.1.3.2.6. Analysis of perfusion CMR
    - 2.1.3.2.7. Comparison with nuclear perfusion tests
    - 2.1.3.2.8. Prognostic information
  - 2.1.3.3. Evaluation of regional & global function during pharmacologic stress
    - 2.1.3.3.1. Protocol/scanning technique
    - 2.1.3.3.2. Indications
    - 2.1.3.3.3. Contraindications
    - 2.1.3.3.4. Clinical validation & application
    - 2.1.3.3.5. Comparison with dobutamine stress echo
    - 2.1.3.3.6. Prognostic information
  - 2.1.3.4. Microvascular dysfunction
- 2.1.4.Coronary imaging
  - 2.1.4.1. Sequences & scanning technique
  - 2.1.4.2. Clinical validation & application
  - 2.1.4.3. Anomalous coronary arteries
  - 2.1.4.4. Coronary aneurysms
  - 2.1.4.5. Coronary vein imaging
- 2.1.5. CMR in Resynchronisation therapy (see also in cardiomyopathy section)
  - 2.1.5.1. CMR methods to assess dyssynchrony and myocardial damage
  - 2.1.5.2. Clinical validation & application
  - 2.1.5.3. Prognosis
- 2.2. Acute Ischemic Heart Disease
  - 2.2.1.Acute coronary syndromes (ACS)
    - 2.2.1.1. Clinical application of CMR in the Emergency department
      - 2.2.1.1.1. Indications
      - 2.2.1.1.2. Scanning protocol
      - 2.2.1.1.3. Detection of ACS
      - 2.2.1.1.4. Differential diagnosis (chest pain, troponin rise & normal coronary arteries)
        - 2.2.1.1.4.1. Myocarditis
        - 2.2.1.1.4.2. Tako-tsubo cardiomyopathy
        - 2.2.1.1.4.3. Myocardial infarction with spontaneous recanalisation
        - 2.2.1.1.4.4. Acute aortic syndromes (Aortic dissection/intramural haematoma/ penetrating atherosclerotic ulcer)
        - 2.2.1.1.4.5. Pulmonary embolism
    - 2.2.1.2. Assessment of myocardial oedema
      - 2.2.1.2.1. T2 weighted CMR (sequences, scanning technique)
      - 2.2.1.2.2. Area at risk & Salvaged area
      - 2.2.1.2.3. Novel techniques for oedema (T2-mapping)
    - 2.2.1.3. Peri-procedural injury

2.2.1.3.1. Clinical & prognostic significance
2.2.2. Complications of acute myocardial infarction
2.2.2.1. Thrombus
2.2.2.2. Pseudoaneurysm – contained myocardial rupture
2.2.2.3. True aneurysm
2.2.2.4. VSD
2.2.2.5. Acute mitral regurgitation
2.2.3. No reflow phenomenon – Microvascular obstruction (MVO)
2.2.3.1. CMR techniques for MVO assessment
2.2.3.2. Early vs Persistent MVO
2.2.3.3. Differentiation from thrombus
2.2.3.4. Clinical validation & application
2.2.3.5. Infarct/reperfusion haemorrhage
2.2.4. Peri-infarct zone
2.2.4.1. Definition
2.2.4.2. Clinical & prognostic significance

3. Myocardial (cardiomyopathies, myocarditis, and non-ischaemic heart disease)
3.1. Hypertrophic cardiomyopathy (HCM)
3.1.1. Various patterns of hypertrophy
3.1.2. RV involvement
5.1.3. Late gadolinium enhancement (LGE) in HCM
5.1.3.1. Frequency
5.1.3.1. Typical spatial distribution
5.1.3.1. Various morphologies
5.1.4. CMR findings and indication for ICD
5.1.5. Mitral valve in HCM
5.1.6. CMR findings in HCM and prognosis
5.1.7. Left atrium and HCM
5.1.8. Relative diagnostic yields for HCM by echo and CMR
5.1.9. Differential diagnosis of LV hypertrophy
5.1.10. Differential diagnosis of LVOT obstruction
5.1.11. Guidelines´ recommendation regarding hypertrophic cardiomyopathy and CMR
3.2. Dilated cardiomyopathy (DCM)
3.2.1. LGE in DCM
3.2.1.1. Frequency
3.2.1.2. Typical spatial distribution
3.2.1.3. Prognostic importance
3.2.2. Relative diagnostic yields for DCM by echo and CMR
3.2.3. Potential of CMR to reveal underlying cause of DCM
3.2.4. Differential diagnosis in heart failure of unclear aetiology
3.3. Takotsubo-Cardiomyopathy (TTC) = broken heart syndrome, stress cardiomyopathy
3.3.1. Definition
3.3.2. Typical patient characteristics
3.3.3. Recommended CMR sequences
3.3.4. Clinical importance of CMR in TTC
3.3.5. Time course
3.4. Arrhythmogenic cardiomyopathy (ARVC)
3.4.1. Role of CMR according to the 2010 task force criteria
3.4.2. Appropriate ways to assess RVEDV and RVEF and wall motion abnormalities
3.4.3. Absence of right heart failure
3.4.4. Importance, pitfalls, challenges of RV LGE
3.4.5. Differential diagnosis of RV enlargement
3.4.6. LV involvement
3.5. Noncompaction cardiomyopathy (NCP)
3.5.1. Criteria for NCP (including differences in echo and CMR criteria)
3.5.2. Relative diagnostic yields for NCP by echo and CMR
3.5.3. LGE in NCP

- 3.6. Myocarditis
  - 3.6.1. LGE in myocarditis
  - 3.6.2. Importance of early imaging
  - 3.6.3. T2-weighted imaging
  - 3.6.4. What does the consensus paper recommend
- 3.7. Transplant Cardiomyopathy
  - 3.7.1. Sequences
  - 3.7.2. Clinical importance of CMR imaging
- 3.8. Cardiac involvement in systemic disease / secondary cardiomyopathies
  - 3.8.1. Vasculitis
    - 3.8.1.1. LV dysfunction
    - 3.8.1.2. LGE pattern
  - 3.8.2. Muscle dystrophy
    - 3.8.2.1. LV enlargement, LV dysfunction
    - 3.8.2.2. LGE pattern
    - 3.8.2.3. Fatty infiltration
  - 3.8.3. Sarcoidosis
    - 3.8.3.1. Patterns of LGE
    - 3.8.3.2. Frequency of LGE
    - 3.8.3.3. Extracardiac findings in sarcoidosis
  - 3.8.4. Amyloidosis
    - 3.8.4.1. LGE-pattern and contrast kinetics
    - 3.8.4.2. Typical cardiac morphology and function
    - 3.8.4.3. Pericardial and pleural effusions
    - 3.8.4.4. Related: Contrast administration in renal insufficiency
    - 3.8.4.5. Relative diagnostic yield of echo and CMR
  - 3.8.5. Iron overload cardiomyopathies
    - 3.8.5.1. Concept and challenges of T2* measurement
    - 3.8.5.2. Location of myocardial T2* measurement
    - 3.8.5.3. hepatic involvement
  - 3.8.6. Athlete's heart
    - 3.8.6.1. Ways to differentiate athlete´s heart from cardiomyopathy
    - 3.8.6.2. Types of sports typically associated with cardiac changes
  - 3.8.7. Endomyocardial fibrosis
    - 3.8.7.1. Restrictive pathophysiology
    - 3.8.7.2. LGE
    - 3.8.7.3. RV involvement
    - 3.8.7.4. Thrombus formation
  - 3.8.8. Chagas' disease
    - 3.8.8.1. Pathophysiology
    - 3.8.8.2. Morphology
    - 3.8.8.3. LGE
    - 3.8.8.4. Epidemiology
  - 3.8.9. Fabry´s disease
    - 3.8.9.1. LVH
    - 3.8.9.2. LGE, replacement fibrosis, spatial distribution
    - 3.8.9.3. Extracardiac findings
    - 3.8.9.4. Epidemiology
    - 3.8.9.5. Key elements of diagnosis
- 3.9. CMR in Resynchronisation therapy (see also in ischemic heart disease section)
  - 3.9.1. CMR methods to assess dyssynchrony and myocardial damage
  - 3.9.2. Clinical validation & application
  - 3.9.3. Prognosis

4. Pericardium
   - 4.1. Normal anatomy
     - 4.1.1. Pericardial layers, sinuses and recesses
     - 4.1.2. Pericardial fluid
     - 4.1.3. Signal characteristics on spin-echo and gradient-echo sequences
   - 4.2. Pathophysiology

- 4.2.1. Role of pericardium in ventricular dynamics
- 4.2.2. Causes of pericardial disease
- 4.2.3. Symptoms and signs of pericardial disease
- 4.2.4. Findings on ECG and on other imaging modalities

4.3. Congenital absence of pericardium

4.4. Pericarditis

- 4.4.1. Etiology
- 4.4.2. Diagnosis
  - 4.4.2.1. Pericardial thickening
  - 4.4.2.2. Signal characteristics

4.5. Pericardial effusion

- 4.5.1. Size
- 4.5.2. Location
- 4.5.3. Fluid characteristics

4.6. Pericardial tamponade

- 4.6.1. Etiology
- 4.6.2. Diagnosis

4.7. Constrictive pericarditis

- 4.7.1. Etiology
- 4.7.2. Diagnosis

4.8. Pericardial cysts

- 4.8.1. Location
- 4.8.2. Signal characteristics
- 4.8.3. Differential diagnosis

4.9. Pericardial tumours (see also section 9.1.4)

- 4.9.1. Primary
  - 4.9.1.1. Benign
  - 4.9.1.2. Malignant
- 4.9.2. Secondary (metastases)

5. Vascular

5.1. Aorta

- 5.1.1. Normal anatomy
  - 5.1.1.1. Segments
  - 5.1.1.2. Size
  - 5.1.1.3. Major branches
    - 5.1.1.3.1. Aortic arch branches (brachiocephalic trunk, carotid, subclavian and vertebral arteries)
    - 5.1.1.3.2. Abdominal side branches (coeliac trunk, mesenteric and renal arteries)
    - 5.1.1.3.3. Aortic terminal branches (iliac arteries, common femoral arteries)
  - 5.1.1.4. Normal variants
- 5.1.2. Physiopathology
  - 5.1.2.1. Role as a conducting vessel
  - 5.1.2.2. Predisposing factors for aortic dilatation and acute aortic syndromes
    - 5.1.2.2.1. Genetic/Congenital
    - 5.1.2.2.2. Acquired
  - 5.1.2.3. Understand appropriate management for each condition
  - 5.1.2.4. MRI sequences
    - 5.1.2.4.1. Spin-echo
    - 5.1.2.4.2. Gradient-echo
      - 5.1.2.4.2.1. Phase contrast flow imaging
      - 5.1.2.4.2.2. Steady state free precession
- 5.1.3. 3D angiography
  - 5.1.3.1.1. Contrast-enhanced
  - 5.1.3.1.2. Steady state free precession
- 5.1.4. Aortic coarctation (see congenital heart disease)
- 5.1.5. Anomalies of the aortic arch (see congenital heart disease)
- 5.1.6. Aortic aneurysm

- 5.1.6.1. Location
- 5.1.6.2. Size and length
- 5.1.7. Aortic dissection
  - 5.1.7.1. Location (Stanford and De Bakey classifications)
  - 5.1.7.2. Extension of intimal flap, entry and re-entry sites
  - 5.1.7.3. Relationship between true and false lumina
  - 5.1.7.4. Complications
- 5.1.8. Intramural haematoma
- 5.1.9. Aortic ulcer
- 5.1.10. Aortitis, infectious and non-infectious

5.2. Pulmonary vessels

- 5.2.1. Normal anatomy
  - 5.2.1.1. Size
  - 5.2.1.2. Segmentation
- 5.2.2. Physiopathology
  - 5.2.2.1. Pulmonary hypertension
  - 5.2.2.2. Pulmonary valve disease
  - 5.2.2.3. Congenital heart disease
    - 5.2.2.3.1. Primary
    - 5.2.2.3.2. Iatrogenic
- 5.2.3. Evaluation of pulmonary vessels
  - 5.2.3.1. Spin-echo sequences
  - 5.2.3.2. Gradient-echo sequences
    - 5.2.3.2.1. Steady state free precession
    - 5.2.3.2.2. Phase contrast flow imaging
    - 5.2.3.2.3. Contrast-enhanced angiography
- 5.2.4. Acquired pulmonary artery disease (for congenital pulmonary artery disease see congenital heart disease)
  - 5.2.4.1. Pulmonary artery hypertension
  - 5.2.4.2. Pulmonary embolism
- 5.2.5. Pulmonary vein disease
  - 5.2.5.1. Anomalous pulmonary venous drainage (see congenital heart disease)
  - 5.2.5.2. Pulmonary vein stenosis
    - 5.2.5.2.1. Compression by surrounding structures
    - 5.2.5.2.2. As a complication of radiofrequency therapy

5.3. Systemic veins

- 5.3.1. Normal anatomy
- 5.3.2. Normal variants
- 5.3.3. Acquired disease
  - 5.3.3.1. Thrombus formation
  - 5.3.3.2. External compression
  - 5.3.3.3. Tumour invasion

6. Valves

6.1. Basic physiological and pathophysiological principals and CMR sequences

- 6.1.1. CMR sequences for analyzing valve morphology, quantifying flow, chambers volumes and function (see also section 1 and 3)
- 6.1.2. Normal valve flow profiles
- 6.1.3. Aetiology of valve stenosis and regurgitation
- 6.1.4. Flow patterns of stenosis and regurgitation
- 6.1.5. Anatomic area, continuity equation and pressure gradients estimation in the assessment of stenosis severity
- 6.1.6. Severity indices for valve regurgitation assessment
- 6.1.7. Impact of valve diseases on heart chamber geometry, volumes, function, mass. Myocardial structure
- 6.1.8. Complementary evaluation of the great vessels
- 6.1.9. Diagnostic accuracy, strengths and weaknesses in comparison with echocardiography, catheterization and CT

6.2. Assessment of Valve Stenosis

- 6.2.1. Assessment of mechanisms and aetiology

- 6.2.2. Flow jet origin and orientation/direction
- 6.2.3. Strengths, difficulties and limitations: Methods of quantification of stenosis severity
- 6.2.4. Specific issues for aortic valve stenosis
  - 6.2.4.1. LVOT assessment
  - 6.2.4.2. Assessment of LV remodeling: volumes, function, wall thickness and mass, LGE fibrosis patterns
  - 6.2.4.3. Methods and clinical impact of diastolic function
  - 6.2.4.4. Detection/significance of associated LVOT obstruction
  - 6.2.4.5. Relation between aortic and pulmonary outflow anatomy for aortic valve implantation (TAVI)
- 6.2.5. Specific issues for sub & supravalvular aortic stenosis
  - 6.2.5.1. Type and localization of stenosis
  - 6.2.5.2. LVOT morphology and size (subvalvular). Aortic root and ascending aorta morphology and size (supravalvular)
  - 6.2.5.3. Differentiation of valve stenosis from sub and supravalvular stenosis
- 6.2.6. Specific issues for mitral valve stenosis
  - 6.2.6.1. Significance of left atrium size and RV remodeling.
  - 6.2.6.2. Left atrial thrombus diagnosis
  - 6.2.6.3. Tricuspid and pulmonary valve function
- 6.2.7. Specific issues for pulmonary stenosis (see Congenital Heart Disease section)
- 6.2.8. Specific issues for tricuspid valve stenosis
  - 6.2.8.1. Significance of right atrium size.
  - 6.2.8.2. Venae cavae dimensions

- 6.3. Assessment of Valve Regurgitation
  - 6.3.1. Assessment of the mechanisms and aetiology
  - 6.3.2. Regurgitant jet origin and orientation/direction
  - 6.3.3. Strengths, difficulties and limitations of the methods of quantification of the regurgitation severity
  - 6.3.4. Specific issues for mitral regurgitation
    - 6.3.4.1. LV geometry, function and late gadolinium enhancement in the mechanism of regurgitation
    - 6.3.4.2. Left atrium size, right heart chambers & valves.
    - 6.3.4.3. CMR findings and selection of patients for intervention or surgery
  - 6.3.5. Specific issues for aortic regurgitation
    - 6.3.5.1. LV remodeling: volumes, function, thickness and mass
    - 6.3.5.2. Importance of the complementary study of aorta
    - 6.3.5.3. CMR findings and selection of patients for surgery
  - 6.3.6. Specific issues for tricuspid regurgitation
    - 6.3.6.1. Significance of right atrium size and RV remodelling
    - 6.3.6.2. Venae cavae dimensions
  - 6.3.7. Specific issues for pulmonary regurgitation
    - 6.3.7.1. Assessment of RVOT, pulmonary artery, RV remodelling
    - 6.3.7.2. CMR findings and selection of patients for intervention
- 6.4. Prosthetic heart valves
  - 6.4.1. Specific morphology and signal characteristics for valve prosthetic annulus, biological and mechanical prosthetic valves
  - 6.4.2. Normal and abnormal SSFP flow patterns
  - 6.4.3. CMR and its clinical role in the evaluation of prosthetic heart valves

7. Masses/Tumors
   - 7.1. Epidemiology, pathology and clinical presentation of each type of mass
     - 7.1.1.Primary tumours
       - 7.1.1.1. Benign, e.g.
         - 7.1.1.1.1. Myxoma
         - 7.1.1.1.2. Lipoma
         - 7.1.1.1.3. Papillary fibroelastoma
         - 7.1.1.1.4. Fibroma

- 7.1.1.1.5. Rhabdomyoma
- 7.1.1.1.6. Haemangioma
- 7.1.1.2. Malignant, e.g.
- 7.1.1.2.1. Sarcomas
- 7.1.1.2.2. Lymphoma
- 7.1.2. Secondary tumours
- 7.1.2.1. Metastasis
- 7.1.3. Pseudotumors: Vein graft aneurysms, caseous mitral annular calcifications, vegetation, haematoma
- 7.1.4. Thrombus
- 7.1.5. Pericardial masses (see also section 6.9)
- 7.1.5.1. Benign, e.g.
- 7.1.5.1.1. Cysts
- 7.1.5.1.2. Lipoma
- 7.1.5.1.3. Teratoma
- 7.1.5.1.4. Fibroma
- 7.1.5.1.5. Haemangioma
- 7.1.5.2. Malignant, e.g.
- 7.1.5.2.1. Mesothelioma
- 7.1.5.2.2. Lymphoma
- 7.1.5.2.3. Sarcoma
- 7.1.5.2.4. Metastatic
- 7.1.6. Other mediastinal masses
- 7.2. Approach to tumours
- 7.2.1. Size
- 7.2.2. Shape
- 7.2.3. Location
- 7.2.4. Signal characteristics on different sequences
- 7.2.4.1. T1 and T2 weighted spin-echo
- 7.2.4.1.1. With fat suppression (vs STIR)
- 7.2.4.1.2. Without fat suppression
- 7.2.4.2. Gradient-echo
- 7.2.4.2.1. Steady state free precession
- 7.2.4.2.2. Fast low-angle shot
- 7.2.4.3. Perfusion
- 7.2.4.4. Early and late after gadolinium injection
- 7.2.5. Obstruction to flow
- 7.2.6. Compression, erosion and invasion of surrounding structures
- 7.3. Features to differentiate benign from malignant masses
- 7.4. Understand advantages and limitations of assessment of masses by CMR
- 7.5. Compare with other imaging modalities

8. Congenital heart disease and adult congenital heart disease
- 8.1. Basics & CMR sequences
- 8.1.1. Important techniques
- 8.1.1.1. MR Ventricular Assessment
- 8.1.1.1.1. Type of sequences – gated vs. real-time imaging
- 8.1.1.1.2. Image planning – short vs. long axis
- 8.1.1.1.3. Segmentation – Inclusion or exclusion of RV trabeculations
- 8.1.1.2. MR Flow Assessment
- 8.1.1.2.1. Type of sequences - free breathing vs. breath hold imaging
- 8.1.1.2.2. Image planning – associated errors
- 8.1.1.2.3. Sources of error
- 8.1.1.3. MR Anatomical Assessment
- 8.1.1.3.1. Type of sequences (3D)
- 8.1.1.3.2. Type of sequences (2D)
- 8.1.2. Important concepts
- 8.1.2.1. Sequential segmental analysis
- 8.1.2.1.1. Atrial situs

8.1.2.1.2. Venous connections
8.1.2.1.3. Ventricular topology
8.1.2.1.4. Atrioventricular connection
8.1.2.1.5. Ventricular arterial connection
8.1.2.2. Congenital causes of dilated ventricles
8.1.2.2.1. Type of causes – valve regurgitation and shunts
8.1.2.2.2. Shunts – calculate Qp:Qs
8.1.2.2.3. Dilated RV – Common congenital causes
8.1.2.2.4. Dilated LV – Common congenital causes
8.1.2.3. Congenital causes of hypertrophied ventricles
8.1.2.3.1. Hypertrophied RV – Common congenital causes
8.1.2.3.2. Hypertrophied LV – Common congenital causes

8.2. Specific lesions
8.2.1.Atria and Venous return
8.2.1.1. ASD
8.2.1.1.1. Types – Understand the differences between Secundum, Sinus venosus, and partial AVSD
8.2.1.1.2. Physiology - Type of shunt, relationship between ventricular and great artery stroke volume
8.2.1.1.3. Other findings – Be aware of additional findings
8.2.1.2. Partial anomalous pulmonary venous drainage (PAPVD)
8.2.1.2.1. Types – different subtypes and their anatomy
8.2.1.2.2. Physiology – Type of shunt, relationship between ventricular and great artery stroke volumes
8.2.1.2.3. Other findings – Be aware of additional findings
8.2.1.3. Anomalous vena caval return
8.2.1.3.1. Types – common abnormalities
8.2.1.3.2. Physiology – Understand which cause symptoms
8.2.1.3.3. Other findings – Be aware additional findings
8.2.2.Atrioventricular valves
8.2.2.1. AVSD
8.2.2.1.1. Types and different nomenclature
8.2.2.1.2. Valvular regurgitation – Know how to assess regurgitation
8.2.2.1.3. Shunting – relationship with type of AVSD
8.2.2.2. Ebstein's anomaly
8.2.2.2.1. Anatomy of tricuspid valve
8.2.2.2.2. Imaging – challenges
8.2.2.2.3. Physiological consequences
8.2.3.Ventricular anomalies +/- other lesions
8.2.3.1. VSD
8.2.3.1.1. Types – Different position of VSD (inlet, outlet, apical)
8.2.3.1.2. Physiology - Type of shunt, relationship between ventricular and great artery stroke volumes
8.2.3.2. Tetralogy of Fallot (unrepaired)
8.2.3.2.1. Findings – know the 4 findings
8.2.3.2.2. Physiology – Type of shunt, relationship with anatomy
8.2.3.3. Tetralogy of Fallot (repaired)
8.2.3.3.1. Findings – know repaired anatomy
8.2.3.3.2. Problems – common post op complications
8.2.3.3.3. Physiology – Understand physiology of complications
8.2.4.Outflow anomalies (see valves chapter)
8.2.4.1. Transposition of the Great Arteries (repaired)
8.2.4.1.1. Findings – repaired anatomy for arterial switch and atrial switch (Senning or Mustard procedures)
8.2.4.1.2. Problems (arterial switch) – understand complications and their physiology
8.2.4.1.3. Problems (atrial switch) – understand complications and their physiology
8.2.4.2. Congenitally corrected transposition of the Great Arteries

- 8.2.4.2.1. Findings – what is the anatomy
- 8.2.4.2.2. Appearance – common misdiagnosis
- 8.2.4.2.3. Other findings – Be aware additional findings

8.2.5. Great vessel anomalies

- 8.2.5.1. Aortic Coarctation
  - 8.2.5.1.1. Types and methods of repair
  - 8.2.5.1.2. Common post-operative/intervention complications
  - 8.2.5.1.3. Physiology - how to assess peak velocity and collateral flow - challenges
  - 8.2.5.1.4. Other findings – Be aware additional findings
- 8.2.5.2. Patent ductus arteriosus
  - 8.2.5.2.1. Physiology – Understand PDA causes L-R shunting and how to assess (volumes and mass)
  - 8.2.5.2.2. Physiology – Type of shunt, relationship between ventricular SV and Pulmonary perfusion
- 8.2.5.3. Vascular Rings
  - 8.2.5.3.1. Findings – Understand that how rings are formed
  - 8.2.5.3.2. Problems – Know which arch and PA abnormalities cause symptoms
- 8.2.5.4. Single ventricles
  - 8.2.5.4.1. Types of palliation – Basic understanding of staged approach to palliation
  - 8.2.5.4.2. Physiology – Understand basic physiology of Fontan circulation

9. Incidental (non-cardiovascular) findings
   - 9.1. Neck (thyroid nodules, adenopathy)
   - 9.2. Thorax
     - 9.2.1. Lung (airspace disease, mass)
     - 9.2.2. Pleura (effusion, neoplasm)
     - 9.2.3. Mediastinum
       - 9.2.3.1. Esophagous (hernia, mass, dilatation/thickening)
       - 9.2.3.2. Solid & cystic masses, adenopathy
     - 9.2.4. Bone (fracture, neoplasm, infection)
     - 9.2.5. Chest wall mass (breast, axilla)
   - 9.3. Abdomen
     - 9.3.1. Liver (cyst/hemangioma, mass, parenchymal disease)
     - 9.3.2. Kidney (cyst, mass, hydronephrosis, parenchymal disease)
     - 9.3.3. Adrenal mass
     - 9.3.4. Spleen (size, lesion)
     - 9.3.5. Other
       - 9.3.5.1. Gallstones/Cholecystitis
       - 9.3.5.2. Ascites
   - 9.4. Management of incidental findings