EUROPEAN ASSOCIATION OF CARDIOVASCULAR IMAGING A Registered Branch of the ESC

EACVI CORE SYLLABUS

A learning framework for continuous medical education in echocardiography

Prepared by the Education Committee of the European Association of Cardiovascular Imaging

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Foreword

The EACVI Core Syllabus aims to encourage homogeneous cardiovascular imaging education in Europe. The European Association of Cardiovascular Imaging is a world leader in education, providing high quality educational opportunities through congresses, journals, website and other educational products.

The Echocardiography Core Syllabus provides a summary of the core knowledge for cardiology fellows, for cardiac sonographers and for continuing medical education of trained cardiologists. The Core Syllabus aims to be an educational framework in teaching hospitals across Europe. It will also be used to standardize the content of educational activities within the European Association of Cardiovascular Imaging and in its external relations with National Societies and National Working Groups on echocardiography.

Preface

Providing adequate education is one of the main goals of the European Association of Cardiovascular Imaging (EACVI). Echocardiography is able to provide an impressive amount of information using different modalities (M-mode, 2D and 3D, Doppler, Doppler derived techniques and speckle tracking echocardiography), approaches (transthoracic-TTE, transesophageal-TOE, intravascular, epicardial), and applications (e.g. stress and contrast echocardiography), but needs adequate teaching and training of operators, performed in a cost effective and reliable way. The purpose of producing a Core Syllabus is to enumerate the elements of knowledge that the EACVI expects the European echocardiography operator to possess. This document represents a driving force for the EACVI to deliver educational resources which to assist the learners in performing comprehensive and accurate echocardiographic studies and achieving accreditation.

Introduction

Echocardiography is an ultrasound based imaging modality which provides a noninvasive assessment of the cardiac structure, function and haemodynamics, generating a real time image. Echocardiography is a major contributor to the practice of cardiology, allowing diagnosis and follow up of cardiovascular disease in children and adults. The ability of echocardiography to provide unique non invasive information with minimal discomfort or risk, without using ionizing radiation, together with its portability, immediate availability, and repeatability, explains its use in all fields of cardiology. However, echocardiography remains an operator dependent technique. Thorough knowledge of cardiovascular anatomy and pathophysiology together with appropriate technical skills are necessary for a comprehensive and clinically useful echocardiographic study to be performed. The required knowledge and skills can only be gained through supervised education and training in an appropriate environment.

At the end of its training, an operator should have the skills to perform a transthoracic and/or transoesophageal echocardiographic examination using the full range of widely used and validated diagnostic capabilities to identify the nature and establish the severity of cardiac diseases in order to guide clinical management of patients. Obtaining diagnostically relevant information by echocardiography requires continuous integration of one's knowledge with the ultrasonographic image content. Knowledge of ultrasound physics and instrumentation and its continuous application during the examination is prerequisite to obtain optimal data.

The Core Syllabus

The EACVI Core Syllabus describes the elements of knowledge needed to perform and report echocardiography studies and to achieve accreditation.

The purposes of the Core Syllabus

The Core Syllabus provides a framework for EACVI to use internally for its educational and accreditation activities and externally for its relations with National Societes and/or Working Groups on echocardiography.

Internal use of the Core Syllabus

The EACVI Core Syllabus will represent a platform to facilitate a structured approach to Continuous Education in echocardiography. The Education Committee will use the document to develop educational opportunities accordingly:

- Teaching courses
- Recommendation documents
- Books, slide sets and other educational materials

External use of the Core Syllabus.

The EACVI Core Syllabus will assist in:

- Educational events planning and content standardization for National Societes/Working Groups
- Cardiovascular imaging education standardization and therefore standardization of cardiovascular diagnosis and management throughout Europe
- Updating the European Society of Cardiology Core Syllabus and Core Curriculum in Clinical Cardiology

European Board of Accreditation in Cardiology (EBAC)

EBAC role is to provide the highest level of quality in international CME throughout Europe. The EACVI Core Syllabus will facilitate the cooperation between EACVI Education Committee, ESC Education Committee and EBAC in providing adequate CME credits for various educational programmes run throughout Europe.

Future developments

The Core Syllabus is the first step in the development of the EACVI Core Curriculum. The Core Curriculum will be an expansion of the Core Syllabus based on educational objectives. It will specify teaching, training and assessment methods.

Acknowledgements

We acknowledge the work of the previous EAE Education Committee in preparing the first Core Syllabus for Echocardiography.

1. GENERAL PRINCIPLES OF ECHOCARDIOGRAPHY

Physics of Ultrasound

1/Sound wave - compression and rarefaction

2/Audible sound versus ultrasound frequency range

3/Diagnostic frequency range - trade-off penetration / spatial resolution

4/Ultrasound wave characteristics

-Frequency - relation to wave length

-Amplitude - relation to Power, Intensity, Pressure

5/Ultrasound propagation

-Average speed in tissues

-Reflection / Refraction / Transmission at interfaces

- Acoustic impedance
- Interface characteristics dependence
- Return signal ratio angle and acoustic impedance, mismatch dependence -Scattering
 - Return signal ratio scattered size and frequency dependence

-Attenuation

- Sources of attenuation
- Frequency dependence
- Effect on images

Transducers

1/Transducer components

-Piezoelectric element

- Piezoelectric effect
- Artificial ceramics
- Resonant frequency
- -Damping material

-Impedance matching layer

2/Current transducers characteristics

-Matrix array

- -Phased array
- -Sound beam formation in 2D and 3D
- -Electronic steering and focusing
- -Focal zone characteristics (maximum intensity, depth of focus, focal area)
- -Focusing on transmitted ultrasound and multiple focusing on received ultrasound
- -Side lobes influence on image quality
- -Transducer frequency range
 - Adjust considering penetration / resolution requirements
 - Adult / paediatric transducers

-Size, shape, aperture (footprint)

Ultrasound Imaging

1/Currently used imaging modes

- -2D current characteristics and applications
- -3D current characteristics and applications
- -Speckle tracking echocardiography
- -Doppler Pulsed Wave, Continuous Wave, Colour Flow, Tissue
- -M-mode current recommendations

2/Digital echocardiography machines

- -Signal processing
- -Binary systems
- -Image storage temporary / permanent (CD, external digital archiving system)
- -DICOM and HL7 principles
- -Pixels effect on image resolution
- -Display devices digital monitors, flat screen
- -Display controls Brightness, Contrast
- -Postprocessing
- -Off line image analysis / reporting
- -Image comparison capabilities at follow-up

3/General image formation principles

-Interdependence of number of beams (scan lines) - sector width - depth - frame rate

- -Limiting factors for image formation speed
 - Lines density
 - Frame rate / volume rate
 - Ultrasound travelling time
 - Sector width (2D) / volume dimensions (3D)
 - Depth
- 4/Image optimization
 - -Output power
 - -Dynamic range
 - -Compress
 - -Gain overall, time gain compensation, lateral gain compensation
 - -Reject
 - -3D on-line / off-line image optimisation
- 5/Harmonic imaging
 - -Principles
 - -Impact on image quality
 - -Use in contrast echocardiography
- 6/Imaging artefacts and pitfalls
 - -Reverberation artefact
 - -Mirror images
 - -Near field clutter
 - -Side lobes
 - -Refraction artefact
 - -Shadowing
 - -Stitching artefact (3D)
 - -Dropout artefact (3D)
 - -Stationary artefacts (3D)
 - -Aliasing (Doppler)
 - -Blooming (Contrast)
 - -Attenuation (Contrast)
 - -Lateral shadowing (Contrast)
 - -Apical dropout (Contrast)
 - -Lateral dropout (Contrast)

Biologic Effects of Ultrasound and Safety

1/Dose measures (Pressure, Intensity, Power and Exposed Area) 2/Factors affecting acoustic exposure, equipment controls 3/Biological / Physical effects -Cavitation -Heating 4/Electrical and mechanical hazards 5/Contrast echo agents hazard

Quality Assurance of Ultrasound Instruments

1/General concepts

- -Need for quality assurance
- -Nature of a quality assurance program

Doppler Echocardiography

1/Physics

- -Doppler Effect (as related to sampling red blood cell movement)
- -Fast Fourier transformation
- -Doppler equation
- -Angle of incidence
- -Colour Doppler Processing
- 2/Spectral Doppler
 - -Pulsed Wave / Continuous Wave Doppler differences, pros and cons
 - -Spectral displays characteristics, derived information
 - -Spectral broadening and artefacts
- 3/Pulsed Wave Doppler
 - -Sample volume(s)
 - -Aliasing, Nyquist frequency limit, Scale
 - -Pulse Repetition Frequency HPRF Doppler
 - -Maximum depth
 - -Baseline position
- 4/Continuous Wave Doppler
 - -High-velocity measurement capability
 - -The role of the stand-alone probe
- 5/Colour Doppler
 - -Sample volume size
 - -Aliasing, Scale
 - -Maximum depth
 - -Baseline position
 - -Colour display characteristics and derived information
 - -Colour Maps, Variance
 - -Postprocessing options

6/Power Doppler principle

7/Doppler tissue imaging principle

2. THE HISTORY OF ECHOCARDIOGRAPHY

Transducers

1/Mechanical transducers

2/Transducers 1D, 1.5D

3/Arrays (linear, phased, annular)

4/Steering and focusing history (lens, curved element, electronic)

5/Fixed focal length (lens focused single element) / variable focal length (annular array, phased array)

6/Single frequency transducers selection (trade-off penetration/ resolution) 7/Historic transducers size and shape

-Large Transducer: better beam characteristics, sharper focus at deeper depth

-Small Transducer: needs smaller acoustic window, easier to handle

Ultrasound Imaging

1/Historic imaging modes

-A-mode, B-mode

-M-mode – historic role / current recommendations

-2D – development history (fundamental imaging / harmonic imaging)

- -Contrast echo imaging modalities development history
 - (High power / low power and intermittent / continuous imaging)
- -3D development history

2/Analogue echocardiography machines

-Analogue signal and scan conversion

- -Display devices oscilloscopes, television monitors
- -TV lines effect on image resolution
- -Display controls Brightness and Contrast
- -Image storage Paper and Video

Measurements and calculations

1/Left ventricular (LV) systolic function assessment

- -Velocity of circumferential fibre shortening: Vcf = [EDD-ESD] : [EDDxET]
 - EDD = end diastolic diameter
 - ESD = end systolic diameter
 - ET = ejection time

3. THE ECHOCARDIOGRAPHIC EXAMINATION

The Echo Exam

1/Image acquisition principles

-Technical considerations

- Appropriate use of equipment controls
- Recognition of technical artefacts
- Recognition of setup errors

-Standard scanning

• Height and weight to calculate body surface area (BSA) for indexed measurements

- ECG
- Respiratory cycle monitoring (constriction, tamponade)
- Oxygen saturation monitoring (in transoesophageal –TOE studies)
- Perpendicular incidence of ultrasound beam on structures
- Standard image orientation
- Standard relation between scan planes (2D / 3D derived 2D / multiplane)
- Standard windows / 2D views
 - Parasternal
 - Long axis (LAX) view of LV
 - Short axis (SAX) views of LV

At the level of the great vessels

At the level of the mitral valve

At the level of the papillary muscles

At the level of the LV apex

Right ventricular (RV) inflow view

- RV outflow view
- Apical
 - 4 Chambers view
 - 2 Chambers view
 - 3 Chambers view (LAX)

Other apical views (5 Chambers, modified 4 Chambers for RV analysis)

- Suprasternal view
- Subcostal views
- Other acoustic windows
- M-Mode / Anatomical M-mode (AMM) acquisition
 - Parasternal (aortic valve & left atrium (LA), mitral valve, LV)
 - Apical window (tricuspid and mitral valve annular plane excursion)
 - Colour M-mode
- 3D acquisition
 - Focused examination

Apical full volume for LV, RV, LA or right atrium (RA) analysis Apical / parasternal for mitral valve (MV) or tricuspid valve (TV) area measurement in valve stenosis

Parasternal 3D zoom for aortic valve (AV) analysis in suspected bicuspid valve or endocarditis

Complete examination

Multiple windows and modes

2/Myocardial segmentation (LV and RV), coronary territories, bull's eye display 3/Principles of echo measurements

-2D echo (current recommendations)

-M-mode (if feasible to align cursor perpendicular to structures)

4/Special Techniques

-Use of contrast agents

-Speckle tracking

-Provocative manoeuvres

Anatomy and Physiology of the Heart and Great Vessels

1/Left Ventricle

-Dimensions, area, volumes

-Wall thickness, mass

-Global and regional systolic function

-Diastolic function

-LV – RV interdependence

2/Right Ventricle

-Dimensions, area, volumes

- -Global systolic function
- -Diastolic function
- -Echo findings in RV volume / pressure overload
- -Moderator band

3/Left Atrium

-Dimensions, area, volumes

- -LA function phasic function (reservoir, conduit and active contraction) 4/Left atrial appendage – morphology and function
- 5/Right atrium

-Dimensions, area, volumes

-RA function - phasic function (reservoir, conduit and active contraction) 6/Interventricular septum (IVS) – morphology and motion

7/Interatrial septum (IAS) - morphology

8/Left ventricular outflow tract (LVOT)

9/Right ventricular outflow tract (RVOT)

10/Pulmonary veins

11/Inferior (IVC) and superior vena cava (SVC)

12/Great vessels

-Aorta

- Aortic annulus
- Sinuses of Valsalva
- Sinotubular junction
- Ascending aorta
- Aortic arch
- Descending thoracic aorta
- Abdominal aorta
- -Pulmonary artery (PA)
 - Main PA (MPA)
 - Bifurcation
 - Right and left PA
 - Ductus Arteriosus Botall
- 13/Coronary sinus
 - -Normal appearance
 - -Causes of dilatation
 - -Differentiation from descending thoracic aorta
- 14/Coronary arteries (when feasible)
 - -Normal appearance
 - -Doppler flow patterns
- 15/Mitral valve apparatus
 - -Leaflets
 - -Commissures
 - -Scallops
 - -Chordae tendinae
 - -Annulus (Normal size, variability throughout cardiac cycle, non-planar shape)
 - -Papillary muscles
- 16/Aortic valve
 - -Cusps, commissures, annulus
 - -Subvalvular space, supravalvular space
- 17/Tricuspid valve
 - -Leaflets (anterior, septal, posterior)
 - -Papillary muscles
 - -Annulus (distance from the mitral valve annulus)
- 18/Pulmonary valve
 - -Cusps, commissures, annulus
 - -Subvalvular space, supravalvular space
- 19/Pericardium

Arrhythmias and Conduction Disturbances

- 1/Generation of regional dyssynergy
- 2/Effect on valve motion
- 3/Effect on Doppler flow velocity pattern

4. THREE-DIMENSIONAL ECHOCARDIOGRAPHY

Instrumentation

1/Fully sampled matrix array transducer (technical aspects)2/Current 3D transducers are smaller (miniaturized circuits), smaller footprint3/The same transducer can be used for 3D or 2D image acquisition4/Harmonics5/Contrast

Advantages and limitations of 3D compared with 2D

1/Advantages

-No need of mental reconstruction of spatial relations between structures / spatial anatomy

-Realistic anatomical views of the heart valves and other cardiac structures

-Findings easy to communicate to non-imaging cardiologists or cardiac surgeons -Allows 3D guided 2D (aligned) delineation of valve areas

-Calculation of volumes and mass without geometric assumptions or foreshortening

-Concomitant dyssynchrony quantification with the assessment of LV volumes / EF 2/Limitations

-Lower temporal resolution

-Spatial resolution / temporal resolution/ data set volume trade-off

-Image quality remains inferior compared with 2D

-Multibeat full volume acquisition prone to stitching artefact

- With breathing (solution: breath-hold)
- With arrhythmia (solution: single beat acquisition)

-Postprocessing of full volumes (rendering) needed for analysis and display

Image acquisition

1/Real time (live) 3D

-Narrow sector

-Zoom

-Colour

2/Full volume

-Multibeat

-Single beat

-Colour

3/Multiplane / x-Plane

-With or without colour and with or without DTI

Image display

1/Cropping

-On-line

-Off-line

2/Rendering

-Volume Rendering

-Surface Rendering (including wire frame display)

-2D Tomographic Slices

Quantitative analysis

1/Dimensions and Areas

-Measured on 3D guided 2D and on volumetric rendered images

-Examples: MV area in mitral stenosis, EROA in mitral regurgitation from colour 3D 2/Volumes and EF

-LV, LA, RA and RV

3/Mass

4/Size of septal defects

5/Advanced measurements

-LV dyssynchrony

-Mitral valve annulus size and shape

5. CONTRAST ECHOCARDIOGRAPHY AND TISSUE HARMONIC IMAGING

Microbubble properties

1/Microbubble characteristics

-Size

-Stability (persistence) determined by

Gas diffusivity

Encapsulation (shell)

2/Ultrasonic properties

-Increased ultrasonic backscatter

- -Backscatter depends on gas compressibility and shell elasticity
- -Non-linear response harmonic frequencies, power Doppler effect
- -Resonant frequency
- 3/Pressure effects
 - -Ambient pressure
 - -Acoustic pressure

4/Safety

Contrast Agents

1/Ideal contrast agent

2/Right heart contrast (non transpulmonary)

- -Agitated normal saline +/- air +/- blood
- -Characteristics
 - Unstable more stable if blood is added at agitation
 - Large, unequal air microbubbles
- -Clinical indications
 - Intracardiac shunt detection
 - Intrapulmonary shunt estimation
 - Enhance tricuspid regurgitation spectral Doppler tracing
- 3/Right and left heart contrast agents (transpulmonary)
 - -Manufactured Sonovue, Optison, Luminity
 - -Characteristics
 - Microbubbles stabilized by encapsulating the gas in a shell
 - Shell composition: phospholipid or albumin
 - Slow diffusing gases (sulphur hexafluoride, perfluorocarbone)
 - -Clinical indications
 - Enhance LV border delineation
 - Enhance spectral Doppler tracings
 - Myocardial Perfusion (research application)
 - -Modalities of contrast administration
 - Bolus
 - Infusion

Contrast imaging modalities

1/Mechanical index

2/Fundamental imaging versus harmonic imaging

3/High power (Power Doppler) versus low power (Pulse Inversion, Power Modulation) imaging

4/Intermittent (triggered) versus continuous (real-time) imaging

5/Contrast destruction/refill analysis (qualitative and quantitative)

6/Signal to noise ration improvement techniques (background subtraction, filtering)

7/Artifacts - see chapter 1

6. DIMENSIONS, VOLUMES AND MASS

Principles

1/Indexed measurements (per BSA)

2/See "Recommendations for chamber quantification" Eur J Echocardiography (2006) 7, 79-108

Dimension measurements

1/Methods

-Parasternal views

-2D frozen end-diastolic / end-systolic frame

-3D derived 2D images

-M-mode – only if cursor is perpendicular to measured structure

-Timing (end-diastole / end-systole)

2/Pitfalls and limitations

-Image quality

-Worse border delineation on frozen frames than on moving 2D image

-Poor RV borders delineation, including IVS right side border

LV Volumes, SV and EF

1/Methods

-Biplane method of discs (modified Simpson's rule) – supported by studies of outcome

-3D full volume analysis – currently recommended, correlates better with CMR

-Single plane area-length – historic value only

-Timing of 2D frozen frame – automatic in 3D analysis

2/Pitfalls and limitations

-Image quality

-Poor endocardial delineation – improved with harmonic imaging +/- contrast

-Endocardial border more difficult to delineate at end-diastole

-Foreshortening on 2D acquisition (overestimates EF) – 3D overcomes this issue

RV Volumes and EF

1/Methods

-3D full volume analysis - correlates well with CMR

-Timing

2/Pitfalls and limitations

-Image quality

-Limited evidence

-Window and image quality not always appropriate

LV mass

1/Methods

-3D full volume analysis

-2D / M-mode calculation

-Papillary muscles are excluded

2/Pitfalls and limitations

-Image quality

-Limited evidence for 3D

-Wide standard deviations for conventional methods

7. DOPPLER DERIVED NON-INVASIVE HAEMODYNAMICS

Basic Principles

1/Laminar versus turbulent flow 2/Flow velocity profiles

Principles of Volume and Flow Measurement

1/Stroke volume

-Stroke volume (SV) = Cross sectional Area (CSA) x Velocity Time Integral (VTI) -Apy of the 4 values = TV least reliable MV pot very reliable

-Any of the 4 valves – TV least reliable, MV not very reliable

-Assumptions

- Circular annulus areas
- Constant areas throughout cardiac cycle
- Area and velocity measured at same level
- Doppler aligned parallel to blood flow
- No beat to beat variation (average over 5 10 beats in atrial fibrillation)

2/Cardiac output (CO)

 $-CO = SV \times heart rate$

3/Shunt calculation

-Pulmonary-to-system flow ratio (Qp/Qs)

4/Regurgitant volume (RV) and regurgitant fraction (RF) calculation

-RV = volume of blood that regurgitates through incompetent valve

-Regurgitant Fraction = Regurgitant Volume / Stroke Volume

Anterograde Intracardiac Flows

1/LV outflow

-Apical 5 chambers or 3 chambers view

- -Sample volume in LVOT, just on LV side of valve, to obtain laminar flow curve
- -Normal values

2/RV outflow

-Parasternal short-axis view

-Sample volume in the RVOT

-Normal values

3/LV inflow

-Apical 4 chambers view

-Sample volume at the level of the MV ring for inflow quantification

-Sample volume at the level of the MV leaflet tips for flow profile (E, A, E/A)

-Normal values

4/Pulmonary venous flow

-Apical 4 chambers view

-Normal values for S, D, S/D, A velocity and duration

5/Descending aorta flow

6/IVC and SVC flow profile – respiratory variation monitoring

7/Hepatic veins flow profile – respiratory variation monitoring

8/Coronary arteries

-LAD, PDA, LCx

-Coronary flow reserve (use of vasodilator agents)

Intracardiac Pressures

1/Bernoulli equation

-Conservation of energy principle (turbulence induced energy loss results in acceleration) 2/Modified Bernoulli equation

 $-P_1 - P_2 = 4 (V_2^2 - V_1^2)$

3/Simplified Bernoulli equation

- $-P_1 P_2 = 4V_2^2$
 - -Assumptions
 - V₁ is negligible
 - Flow through a stenotic orifice
 - (Not valid for prosthesis or large orifices, e.g. severe tricuspid regurgitation)
 - -Pitfalls and limitations
 - Improper Doppler alignment
 - Poorly recorded signals
 - Failure to detect an eccentric high-velocity jet
 - Long, tubular stenoses (viscous friction component becomes significant)
 - Changes in viscosity (e.g. anemia, polycythemia)
 - V₁ significant
 - Mild stenosis
 - Coexistent regurgitation
 - High CO
 - Prosthetic valves with normal function
 - Pressure recovery
 - Reduced poststenotic turbulence allows recovery of potential energy
 - Echo gradient higher than catheter measurement instantaneous / peak to peak
 - Occurs in mild stenosis, narrow poststenotic vessels, small prosthesis
 -Applications
 - Valvular aortic, pulmonic stenosis
 - Subvalvular aortic, pulmonic stenosis
 - Right ventricular or pulmonary artery systolic pressure
 - $4(V_{TR})^2$ + RAP (RAP estimated from IVC diameter and respiratory variation)
 - Pulmonary artery diastolic pressure $4(V_{endPR})^2 + RAP$
 - Pulmonary artery mean pressure
 - $4(V_{earlyPR})^2 + RAP$
 - LV diastolic pressure

Continuity Equation

1/Conservation of mass principle

-Flow volume before a valve equals flow volume across a vale

2/Equation

-(Area₁) (VTI₁) = (Area₂) (VTI₂)

3/Application to AV functional area calculation

-Technique

-Pitfalls and limitations

- Inaccurate LVOT diameter measurement e.g. due to extensive calcification
- Inaccurate LVOT velocity recording (V1)
- Inaccurate transvalvular velocity recording (V₂ or V_{max})
- Irregular rhythm (e.g. atrial fibrillation average over at least 5 beats)
- Low CO states
- The functional (effective) area differs from the anatomical area (planimetry)

4/Application to MV area estimation

-Technique

-Pitfalls and limitations

• As above + as described at MV SV

Pressure Half-Time method for MV area calculation in mitral stenosis (MS)

1/Definition of Pressure Half-Time (PHT)

2/Determinant factors

- -MV area (MVA)
- -LA pressure

-LV compliance (important mainly in calcific mitral stenosis – older age, less compliant LV)

-LA compliance

-Exclude A velocity in SR

3/Equation

-MV area = 220/PHT (220 = empirical constant)

-Pitfalls and limitations

- Suboptimal Doppler envelope follow the mid-diastolic slope
- LV hypertrophy / diastolic dysfunction
- Coexistence of AR (shortens PHT and overestimates MVA)
- Atrial septal defect
- Immediately after balloon valvotomy changes LA compliance
- Low flow low pressure state
- Arrhythmia

Proximal Isovelocity Surface Area (PISA) for assessment of effective orifice area (EOA)

1/Definition: Flow converges toward a restrictive orifice remaining laminar and forming isovelocity surfaces that approximate hemispheres

2/Conservation of mass principle: *Flow across any isovelocity surface = Flow through orifice*

3/Mitral regurgitation (MR) assessment

- -Flow_{MR} = Area_{shell} x V_{shell} = $2 \prod r^2 x V_r$
 - Flow_{MR} = instantaneous flow rate (ml/s)
 - r = radius of the isovelocity shell (cm)
 - V_r = flow velocity at radius r (cm/s)
- -Effective regurgitant orifice area (EROA)
 - EROA_{MR} = (Flow_{MR}) \div V_{MR}
 - Average effective area of the regurgitant orifice
 - Can grade the severity of regurgitation
- -Regurgitant volume (RV)
 - RVmr = EROmr = EROmr x TVImr

-Assumptions

- -Advantages
 - Can be used with coexistent AR
 - Quantitative assessment

-Limitations

- Multiple jets
- Jet constriction
- Assumption of spherical flow convergence area
- Assumption of a circular regurgitant orifice
- Geometry of isovelocity shells changes with flow rate and pressure gradient
- Phasic changes during the cardiac cycle
- Overestimates EROA in flail leaflet with funnel-shaped convergence zone
- PISA radius measurement not always feasible
- Overestimation of flow rate due to high wall filter increasing Doppler velocities

4/Mitral stenosis assessment

-MV Area = Flow $_{mitral} \div V_{peak inflow}$

-Inverted colour scale

-Advantages

- Can be used with coexistent AR
- MR does not affect MV area calculation

-Limitations

- Same as above
- Less evidence
- Underestimates MV area with higher aliasing velocity (>25 cm/s)

5/Other uses

-Aortic regurgitation

-Tricuspid regurgitation

Estimation of myocardial contractility based on dP/dt

 $1/Approximates\ dp/dt_{max}$ measuring the time taken for MR pressure rise between 1 and 3 m/s

2/Relatively afterload-independent

3/Assumptions

-CW Doppler MR velocity reflects instantaneous LV / LA peak gradient

-LA compliant (LA pressure stable during pre-ejection period)

4/Technique

5/Values

6/Pitfalls and limitations

-Poor MR / Doppler alignment (underestimates)

- -Acute MR (noncompliant LA, LA pressure rise with MR)
- -Preload-dependent

-Poorly reproducible

7/RV myocardial contractility - TR dP/dt - limited evidence

8. ASSESSMENT OF SYSTOLIC FUNCTION

Principles

1/Cardiac cycle - systole, diastole
2/Ejection / ejection time
3/Systolic function

LV performance factors

1/Contractility (inotropy)

-Myocardial fibre shortening velocity

Difficult to assess

Preload and afterload dependent

-LV end-systolic elastance $E_{ES} = P_{ES} / (V_{ES} - V_0)$

- Where P_{ES} is end-systolic pressure, V_{ES} is end-systolic volume, and V_0 is Volume at 0 load

Assessed invasively from pressure/volume loops at different loading conditions

Preload and afterload approximately independent

2/Preload (myocardial fibre length at onset of contraction)

-Assessed by measuring EDV (end diastolic volume) and EDP (end diastolic pressure)

-Higher preload => higher myocardial work, higher stroke volume (*Frank-Starling law*)

3/Afterload (force opposing contraction / myocardial fibre shortening)

-Dependent on

- LV volume and pressure
- Aortic impedance and arterial resistance
- Blood volume and viscosity

4/Ventricular geometry

Global LV Systolic Function

1/Echocardiographic measures of systolic function

- -Ejection fraction (EF)
- -Fractional shortening

-MV annulus displacement / velocity

- -Velocity of circumferential fibre shortening
- -SV and CO
- -Systolic time intervals
- -dp/dt (rate of ventricular pressure rise)
- -Acceleration time
- -Myocardial strain
- 2/Further echocardiographic parameters influenced by systolic function
 - -LV sphericity
 - -LVOT VTI
- 3/LV EF

-Methods

- Visual estimation
 - Well approximated by experienced operators
 - High interobserver variability
- Quantitative assessment EF = ([EDV-ESV] : EDV) x100%
 - ${\scriptstyle \circ}$ 3D (no LV shape assumption, no foreshortening, compares well with CMR)
 - 2D biplane
 - Add contrast for better endocardial delineation
- -Conditions requiring EF interpretation caution (afterload and preload dependent)
 - MR / aortic regurgitation (AR)
 - Aortic stenosis (AS) / severe hypertrophy
 - MS
 - Ventricular septal defect (VSD)
 - Severe anaemia, hyperthyroidism and other hyperdynamic states
 - LV underfilling
- 4/Fractional shortening (%)
 - -Parasternal 2D or M-mode (FS = [EDD-ESD] : EDV)
 - -Historic value

Regional LV Systolic Function

- 1/Myocardial segmentation
 - -16 or 17 segment model
 - -Coronary territories of distribution
- 2/Wall motion analysis
 - -Qualitative
 - Endocardial motion with concomitent myocardial thickening
 - Scar recognition
 - Classification: Hyper-, Normo-, Hypo-, A-, Dyskinesis, Aneurysm
 - -Semi-quantitative
 - Wall motion score index (same score for Hyper- and Normo-)
 - -Quantitative
 - Strain imaging (2D strain, DTI strain)

3/Regional contraction timing analysis

DTI derived techniques timing of systolic events – very high temporal resolution 3D analysis of regional LV volume curves

LV – RV interdependence

1/Pressure and / or volume changes in one ventricle affect the function of the other -Interventricular septum and circumferential myofibres belong to both ventricles

-Pericardium constrains both ventricles within its space

2/Eccentricity index – in either volume or pressure RV overload 3/RV volume overload

-IVS flattening and leftward displacement in diastole only

-Examples: atrial septal defect (ASD), anomalous pulmonary venous drainage, TR 4/RV pressure overload

-IVS flattening and leftward displacement in both systole and diastole

-Examples: cor pulmonale, primary pulmonary hypertension

5/Constrictive pericarditis

Global RV Systolic Function

1/3D EF calculation 2/TV annular plane systolic excursion (TAPSE) 3/RV dp/dt – limited evidence

9. ASSESSMENT OF DIASTOLIC FUNCTION

Principles

1/Diastolic phases: isovolumic relaxation, early diastolic filling, diastasis, late diastolic filling

2/Diastolic parameters: relaxation (active) and compliance (passive)

3/Crossover LV diastolic function / LV filling pressure

Doppler Assessment

1/Measured parameters

-Mitral E and A velocities, E/A ratio, A wave duration

-Isovolumic relaxation time (IVRT)

-Mitral E wave deceleration time, deceleration slope

-Pulmonary venous flow (S/D, A amplitude, A duration)

-Doppler tissue annular early diastolic velocity (e', E/e')

-Colour M-mode flow propagation velocity (Vp)

2/Measurement techniques

3/Diastolic filling patterns / grades of diastolic dysfunction – diagnosis & differential diagnosis

-Normal

-Impaired relaxation (grade I – mild diastolic dysfunction)

-Pseudonormal filling (grade II – moderate diastolic dysfunction)

-Restrictive filling (grade III – severe diastolic dysfunction)

4/Non-invasive estimate of LV filling pressure

5/Diastolic function parameters influencing factors

-Sample volume location, Doppler alignment

-Respiration, Valsalva manoeuvre

-Heart rate, rhythm

-Preload, afterload, exercise

-LV systolic function and ESV

-Atrial function, volume and compliance

-MS, MR, AR

-e' decreased (longitunidal dysfunction of the wall, mitral valve annular

calcification, stenosis, prosthesis, repair ring)

-e' increased in MR (moderate to severe)

6/Clinical application (conditions associated with diastolic dysfunction) **10. DOPPLER TISSUE IMAGING AND SPECKLE TRACKING DERIVED INFORMATION**

Techniques

1/Doppler Myocardial Imaging

-Principles

- Pulsed wave tissue velocity (filtering!)
- Colour coded tissue velocity offline derived velocity curve (frame rate!)
- -Postprocessing
 - Displacement imaging
 - Strain and strain rate
 - Definition
 - Direction of deformation
 - Derived Parameters (Phase, Timing of velocity/strain peaks, etc)
- -Quantitative analysis detailed analysis of regional function
- -Twist, rotation, torsion
- 2/Speckle Tracking Echocardiography
 - -Principles
 - Physical origin of speckles
 - Tracking of speckle motion (frame rate!)
 - -Postprocessing
 - 2D / 3D application
 - Displacement imaging
 - Strain and strain rate
 - Twist, rotation, torsion
 - -Quantitative analysis

3/Limitations /Advantages / Disadvantages of both approaches

Clinical applications

- 1/Haemodynamic assessment
 - -Assessment of filling pressures
- 2/Systolic (global and regional) function / Diastolic function
- 3/Intraventricular dyssynchrony
- 4/Myocardial viability
- 5/Detection of subclinical myocardial dysfunction
 - -(diabetes, obesity, hypertension, valve diseases, cardiomyopathies, coronary disease, etc)

11. CORONARY HEART DISEASE

Coronary anatomy and function

1/Coronary territories of distribution (See chapter 8)
2/Normal and anomalous origin and course
3/Coronary aneurysms
4/Coronary fistulae
5/Normal coronary sinus and malformations
6/Coronary atherosclerosis

Myocardial Ischemia

- 1/Pathophysiology
 - -Ischemic cascade
 - -Coronary perfusion / wall motion relation
- 2/Detection of ischemia
 - -Reduced endocardial motion

- -Reduced thickening/shortening
- -Delayed thickening/shortening
- -Post-systolic shortening
- -Diastolic function changes
- -Quantitative assessment (Strain/Strain Rate Imaging)
- 3/Confounding factors
 - -Translational motion
 - -Tethering by adjacent segments
 - -Scanning plane change
 - -Conduction or pacing abnormalities
- 4/Role of Stress testing (see stress echo)

Myocardial Infarction

1/Diagnostic features

- -Regional wall motion abnormalities
- -Relation between transmurality and regional function
- -Acute vs late phase of myocardial infarction
 - Hypercontractility of non-infarcted segments
 - Diastolic wall thickness
 - Scar
- 2/Diagnosis of complications
 - -Acute ischemic MR
 - -Free wall rupture
 - -Interventricular septal rupture
 - -Aneurysm, pseudoaneurysm
 - -Papillary muscle dysfunction / rupture
 - -RV infarction
 - -LV thrombus
 - -Infarct expansion and extension
 - -LV systolic dysfunction
 - -Pericardial effusion
 - -Intraventricular obstruction in hyperdynamic state

3/Follow-up

- -Remodelling (infarct expansion, global dilatation)
- -Recovery of function
- -LV thrombus
- -Ischemic MR

12. HEART VALVE DISEASES

Principles of valve stenosis assessment

- 1/Fluid dynamics
 - -Antegrade intracardiac flows see chapter 7
 - -Intracardiac pressures see chapter 7
 - Bernoulli equation
 - Modified Bernoulli equation
 - Simplified Bernoulli equation
 - -Continuity equation see chapter 7

2/Factors affecting valve stenosis assessment

- -Physiologic
 - Valve calcification interferes with valve planimetry
 - Low stroke volume
 - Hyperdynamic circulation (e.g. anaemia)

- -Technical
 - Gain
 - Image plane
 - Doppler alignment
- 3/Diagnosis of valve stenosis
 - -Indirect methods
 - Valve anatomy
 - Chamber remodelling (ventricular hypertrophy / atrial dilatation)
 - -Direct methods
 - Prevalvular colour flow acceleration
 - Doppler assessment
- 4/Quantitation of stenosis severity
 - -Integrative approach
 - -Anatomic valve area / calculated functional valve area

Aortic stenosis

1/Aetiology

- -Congenital
 - Bicuspid aortic valve
 - Dysplastic aortic valve
 - Association to Coarctation
- -Rheumatic (suggested by commissural fusion)
- -Degenerative (calcific)

2/Quantitation

-Pressure gradients

- -Valve Area
 - Functional valve area continuity equation using Doppler or 3D derived SV
 - Anatomical valve area planimetry
- -Velocity ratio
- 3/Pitfalls
 - -Low flow aortic stenosis
 - Reduced systolic function => low stroke volume gradient underestimates severity
 - -Paradoxical low flow aortic stenosis
 - Normal systolic function but small cavity => low stroke volume => low gradient
 - -LV function overestimation due to hypertrophy
 - -Pressure recovery
 - -Coexistent moderate or severe MR
 - -Valve calcification makes planimetry difficult
 - -Planimetry to be performed at maximum valve opening
 - -LVOT diameter measurement error is squared in valve area calculation
 - -LVOT diameter measured in 2D TTE / 3D TOE

4/LV remodelling

- -Concentric / eccentric hypertrophy
- -Increase in LV mass

5/Aortic root dilatation, assessment of aortic root, assessment of LVOT 6/Role of stress echo

-Low dose dobutamine or exercise echo for low flow / paradoxical low flow aortic stenosis

-Exercise echo in asymptomatic patients with severe aortic stenosis

-Exercise echo in symptomatic patients with moderate aortic stenosis

Pulmonic Stenosis

1/Aetiology

2/Quantitation

-Pressure gradients

-Functional valve area - continuity equation (RVOT diameter - difficult to measure correctly)

3/Right ventricular remodelling (hypertrophy)

Sub- / Supra-valvular stenosis

1/Aetiology, types

2/Diagnosis and differential diagnosis

- -Inspection of the valve and of the sub- and supra-valvular area
- -Colour Doppler aliasing level
- -HPRF helps localise stenosis level if the velocity is not too high
- -Pressure gradients

Mitral Stenosis

1/Aetiology

- -Rheumatic
- -Mitral annular calcification extending to leaflets
- -Congenital
- -Miscellaneous
 - Myxoma, other tumours
 - Cor triatriatum

2/2D / 3D findings specific for rheumatic stenosis

- -Leaflet thickening, especially at the tips => "doming"
- -Commissural fusion "fish mouth" appearance in SAX
- -Funnel shaped orifice
- -Calcification (leaflets, commissures, chordae, annulus, papillary muscles)
- -Chordal thickening, fusion
- 3/2D / 3D findings specific for calcific stenosis
 - -Calcification of the ring extending to the basal part of leaflets
 - -Free leaflet tips
 - -Lack of commissural fusion (smile shaped orifice)
 - -Pseudo-stenosis differential diagnosis (E smaller than A in MV inflow)
- 4/Quantitation
 - -Mean pressure gradient
 - -Mitral valve area
 - Planimetry (2D and 3D guided 2D)
 - Pressure half-time method
 - Continuity equation
 - PISA method

-Technical considerations and pitfalls for each method 5/Coexistent findings

-Remodelling of cardiac chambers (LA enlargement)

- -Pulmonary hypertension and consequent RV remodelling
- -Thrombi in left atrium and left atrial appendage (TOE)

6/Role of stress echo

-Exercise echo (dobutamine if the patient cannot exercise)

In symptomatic patients with mild to moderate stenosis

-Exercise echo in asymptomatic patients with significant stenosis -Significant findings

- Rise in mean transmitral gradient (to >15mmHg)
- Rise in pulmonary artery systolic pressure (to >60mmHg)

7/Role of echo in percutaneous mitral balloon valvotomy

-Patient selection (suitability for procedure)

- Wilkins score
- Assessment of anatomy and function
- -Echo guidance during valvotomy (role of 3D echo)
 - Transseptal puncture, balloon positioning
 - Immediate assessment of results / complications

8/Indications for TEE

Tricuspid Stenosis

1/Aetiology

- -Rheumatic
- -Congenital
- -Masses obstructing flow (e.g. myxoma, metastatic tumours, thrombus)

-Device lead impairing valve function

2/Quantitation

-Mean pressure gradient

- -Tricuspid valve area
 - Continuity equation
 - Planimetry (3D)
- -Technical considerations and pitfalls of each method

3/Differential diagnosis

4/Other cardiac abnormalities

Principles of valve regurgitation assessment

1/Fluid dynamics

-Regurgitant orifice (size, shape)

-Proximal flow acceleration

- Flow convergence
- PISA method to calculate EROA
- -Vena contracta
 - Laminar flow
 - Area = EROA
- -Flow turbulence in the low-pressure receiving chamber

-Increased antegrade flow volume through the valve

2/Factors affecting regurgitant jet size and shape

-Physiologic

- Regurgitant volume
- Driving pressure
- Size and shape of regurgitant orifice
- Receiving chamber constraint
- Wall impingement
- Timing relative to cardiac cycle
- Influence of coexisting jets or flowstreams

-Technical

- Gain
- Pulse repetition frequency
- Transducer frequency
- Frame rate
- Image plane
- Depth

3/Diagnosis of valve regurgitation

- -Indirect methods
 - Valve anatomy
 - Chamber dilatation and function

-Direct methods

- PW Doppler
- CW Doppler
- Colour flow mapping

4/Valvular regurgitation in normal individuals

5/Quantitation of regurgitation severity

-Semiquantitation

- Colour flow mapping
- CW Doppler signal intensity
- Distal flow reversals

-Quantitative

- Flow Volume at 2 sites
- EROA by PISA

Aortic regurgitation

1/Aetiology

-Cusp abnormalities

- Congenital (bicuspid, quadricuspid and other dysplastic)
- Degenerative (fibrosis / calcification / cusp restriction)
- Rheumatic valve disease
- Cusp prolapse
- Endocarditis
- Drug induced
- Radiation therapy induced
- Miscellaneous (e.g. trauma)

-Aortic root abnormalities

Annuloaortic ectasia (dilatation of sinuses of Valsalva and sino-tubular junction)

Marfan syndrome or other connective tissue disease

Aortic dissection

Dilatation of the aorta in hypertension and coexistent with bicuspid aortic valve

- Miscellaneous (e.g. trauma)
- 2/Functional classification

-Normal leaflet motion

- -Excessive leaflet motion
- -Restricted leaflet motion

3/Consequences

-LV dilatation and hypertrophy (eccentric remodelling – typical appearance)

-High LV mass

-LV hyperdynamic (volume overload)

-Anterior MV leaflet fluttering and/or septal fluttering in eccentric AR (not a sign of severity)

-"Reverse doming" of anterior MV leaflet (posteriorly displaced)

-Jet lesion on septum or anterior MV leaflet

-Premature AV opening

-Increased LVOT velocity

-Diastolic MR

4/Severity assessment

- -Qualitative
 - Morphology assessment

Limitations

 Colour flow Doppler - jet length, width, area, jet width / LVOT width ratio Limitations

- CW Doppler signal intensity and envelope shape
 Limitations
- Diastolic flow reversal in descending thoracic and abdominal aorta Conditions and Limitations
- Diastolic MR

Low sensitivity in chronic AR

Premature MV closure

Low sensitivity in chronic AR, low specificity

-Semiquantitative

- PHT and CW Doppler deceleration slope
 - Conditions and Limitations

Poorly delineated in eccentric jets

- Influenced by LV and aortic compliance, SV, afterload and MS
- Vena contracta
 - Limitations

-Quantitative

- PISA method
 - Limited validation / use compared with MR
- -Regurgitant volume

Limitations

5/Follow- up and surgical timing

-Severity of aortic regurgitation

- -LV ESV / ESD
- -LV EDV / EDD

-LV EF

-Rate of ESV/ESD increase and of EF decrease over time

Mitral regurgitation

1/Aetiology

-Primary

- Myxomatous degeneration (prolapse, flail leaflet, Barlow type valve)
- Ruptured chordae (myxomatous degeneration or trauma)
 - Infective endocarditis
- Mitral annular calcification (MAC)
- Rheumatic
- Löffler's
- Connective tissue disease
- Congenital
- Drug induced
- Radiation therapy induced

-Secondary

- Ischaemic
 - Ischaemic dysfunction of papillary muscle and adjacent myocardial segment
 - Scar involving subvalvular apparatus
 - Ischaemic cardiomyopathy with LV dilatation and leaflet tethering
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- LA dilatation

2/Mechanisms

-Carpentier functional classification

- Type I normal leaflet motion
- Type II excessive leaflet motion
- Type III restricted leaflet motion

-Specific mechanisms

- Annular dilatation
- Elongated and/or ruptured chordae
- Abnormal LV shape/geometry and papillary muscle orientation deforming the
- MV
- Increased rigidity of leaflets
- -Jet direction as a clue to mechanism

3/Consequences

- -LV dilatation and hyperdynamic contraction
- -LA pressure rise
- -LA dilation
- -Rise in systolic pulmonary artery pressure, RA dilatation, and TR
- 4/Diagnosis
 - -2D / 3D morphology analysis
 - Leaflets
 - Leaflet thickenning / redundancy / tethering / retraction / calcification
 - Detection of prolapse / determination of prolapsing scallop
 - Differential diagnosis: prolapse / flail
 - Annulus
 - Dilatation
 - Calcification
 - Subvalvular apparatus
 - Chords elongation / rupture
 - Papillary muscle displacement
 - Rupture of papillary muscle body / papillary muscle head
 - LV walls

5/Timing of regurgitation

-Colour flow M-mode

- 6/Severity assessment
 - -Qualitative
 - MV morphology
 - Limitations
 - Colour flow assessment
 - Limitations
 - CW Doppler signal intensity and shape
 Limitations
 - Flow convergence zone
 - Limitations
 - Systolic flow reversal in pulmonary veins
 - Limitations
 - -Semiquantitative
 - Increased antegrade velocity (E wave) due to increased transmitral flow volume

Limitations

- MV VTI / AV VTI
 - Limitations
- Vena contracta diameter (2D) / EROA (3D)
 - Limitations
- -Quantitative
 - Regurgitant volume (RegV)
 - Limitations
 - PISA (2D and 3D) Instantaneous flow / EROA / RegV
 - Limitations

7/Chronic / acute MR differential diagnosis

8/MV repair feasibility assessment and role in surgical timing -High likelihood of repair

- Posterior leaflet prolapse or ruptured chordae
 - (Especially central posterior scallop P2)
- Small perforation
- -Intermediate likelihood of repair
 - Extensive valve prolapse (multiscallop, bileaflet)
 - Rheumatic
- -Low likelihood of repair
 - Commissural prolapsed
 - Valve calcification
 - Annulus calcification
 - Ischaemic
- 9/Role of stress echo exercise echo

-To evaluate MR severity changes, pulmonary pressure changes and contractile reserve

Tricuspid regurgitation

1/Aetiology

-Primary

- Myxomatous degeneration prolapse / flail leaflet
- Rheumatic
- Carcinoid
- Congenital (e.g. Ebstein's anomaly)
- Endocarditis
- Trauma
- Device leads interfering with valve function
- Marfan syndrome
- Drug induced
- Radiation therapy induced
- -Secondary
 - Secondary to RV dilatation / pulmonary hypertension
- 2/Differential diagnosis primary / secondary TR
 - -Ring dilatation
 - -Morphologic assessment of valve leaflets and subvalvular apparatus
- 3/Consequences
 - -RV and RA dilation
 - -RV volume overload
 - -Vena cava / hepatic veins dilatation
- 4/Timing of regurgitation

-Colour flow M-mode

- 5/Severity assessment
 - -Qualitative
 - TV morphology
 - Limitations
 - Colour flow assessment Limitations
 - CW Doppler signal intensity and envelope shape Limitations
 - -Semiguantitative
 - Vena contracta diameter (2D) / EROA (3D) Limitations
 - Systolic flow reversal in hepatic veins Limitations

- PISA radius
 - Limitations
- Tricuspid inflow
 - Limitations

6/TR based calculation of SPAP not valid in severe torrential ("free"/"massive") TR

Pulmonic Regurgitation

1/Aetiology

-Primary

- Congenital disease (prolapse, hypoplasia)
- Endocarditis
- Carcinoid
- Rheumatic
- -Secondary
 - Post valvuloplasty
 - Post-repair of Tetralogy of Fallot
- 2/Severity assessment
 - -Qualitative
 - Morphology
 - Colour flow PR jet width
 - CW signal intensity and envelope shape
 - Pulmonic: Aortic PW Flow
 - Diastolic flow reversal in MPA
 - -Semiquantitative
 - Vena contracta width
 - -Quantitative
 - PISA determined EROA
 - RegV
- 3/Clinical implications
 - -Decision-making in congenital heart disease
 - -Calculation of diastolic PA pressure

Prosthetic Valves

- 1/Classification
 - -Biologic
 - Stented
 - Porcine xenograft
 - Pericardial xenograft
 - Stentless
 - Porcine xenograft
 - Pericardial xenograft
 - Homograft (allograft)
 - Autograft (Ross procedure)
 - -Mechanical
 - Bileaflet
 - Tilting disk
 - Ball in a cage no longer implanted currently, but still encountered
- 2/Normal findings
 - -Antegrade flow patterns
 - -Physiologic regurgitation
 - -Prosthetic valve "clicks" (mechanical)
- 3/Dysfunction
 - -Stenosis / Obstruction (degeneration, thrombosis, pannus, vegetation)
 - Increased transvalvular velocity compared with previous study
 - Decreased calculated functional valve area

-Paravalvular regurgitation – always abnormal

Paravalvular colour Doppler signal or / and solution of continuation

- -Transvalvular regurgitation significance dependent on type of prosthesis
 - Mechanical thrombosis, pannus, vegetation
 - Increased CW Doppler signal intensity compared with physiologic regurgitation
 - Flow convergence on LV side of MV can be the only clue in mechanical MV • Biologic – degeneration, endocarditis
- -Dehiscence

-Patient prosthesis mismatch

Low calculated functional valve area for BSA

4/Consequences of dysfunction

-Progressive chamber dilation

- LV in AR, MR
- RV in MS (RV dilatation in MV obstruction / thrombosis)

-Pulmonary hypertension

5/Follow-up and the role of baseline assessment

6/Pitfalls and artifacts

-Acousting shadowing

-Reverberations

-Overestimation of transvalvular pressure gradients

-Pressure recovery phenomenon (especially in small size bileaflet mechanical prosthesis)

Endocarditis

1/Diagnostic findings

-Vegetations

- Mass of irregular shape, pedunculated or sessile
- Independent fluttering motion
- Location
 - On the side of the valve hit by the blood stream
 - Secondary jet lesions
 - Pacemaker wires
 - Unusual locations

Chordae tendinae Mural endocardium Eustachian valve Calcified mitral annulus

- Size and risk of embolism
- Differential diagnosis
 - Non-bacterial endocarditis
 - Ruptured chordae
 - Focal, nonspecific thickening or calcium deposits
 - Retained MV leaflets/apparatus after MV replacement
 - Lambl's excrescences and valve "strands"
 - Sutures, strands on prosthetic sewing rings
 - Tumors, thrombi
- -Paravalvular abscess
- -Pseudoaneurysm
- -Perforation / flail leaflet or cusp
- -Intracardiac fistula
- -Valve aneurysm
- -Prosthetic valve dehiscence / new paravalvular regurgitation
- -Healed / chronic vegetations

2/Diagnostic accuracy

-Transthoracic 2D / 3D

-Transesophageal 2D / 3D

(Mandatory except when low clinical suspicion and negative good quality TTE) 3/Surgical indications, timing and intraoperative guidance

4/Clinical indications to perform / repeat TTE / TOE

Valvular Disease in Connective Tissue Disease

1/Systemic lupus erythematosus

-Anatomic and functional abnormalities

-Valve disease severity does not correlate with clinical features severity

-Echo findings

-Leaflet thickening (fibrosis)

Vegetations (Libman-Sacks)

- Valve regurgitation
- Valve stenosis (rare)

2/Ankylosing spondylitis

-Nonspecific thickening of aortic and mitral leaflets

-Thickening of base of anterior mitral leaflet ("subaortic bump")

-Increased echogenicity of posterior aortic wall

-Aortic root dilatation

3/See also Chapter 21

13. CARDIOMYOPATHIES

Dilated cardiomyopathy

1/Diagnostic findings

- -LV dilatation
- -Reduced LV systolic function
- -LV wall thinning
- 2/Associated findings

-LV spherical remodelling

-LV systolic dyssynchrony

-MR

-LV thrombus / spontaneous echo contrast

-RV dilatation

-Pulmonary Hypertension

3/Prognostic role of echo

-Severity of systolic dysfunction

-LV filling pattern

-Coexistence of RV dysfunction

-Severity of LV dilatation

-Systolic dyssynchrony

4/Role in CRT

-LV EF (2D Simpson's biplane, 3D)

- -Dyssynchrony study correct technique and further evidence needed
 - Visual (apical rocking)
 - Quantitative
 - Interventricular dyssynchrony
 - Atrioventricular dyssynchrony
 - Intraventricular dyssynchrony

-Stress echo for viability and dysynchrony assessment

-CRT optimization

Hypertrophic cardiomyopathy

1/Classification

-Familial

- Familial unknown gene
- Sarcomeric protein mutations
- Glycogen storage disease
- Anderson-Fabry
- Disorders of fatty acid metabolism
- Mithocondrial cytopathies
- Syndromic HCM
- -Non-familial
 - Obesity
 - Infants of diabetic mothers
 - Athletic training
 - Amyloid

2/Diagnosis

-Varied patterns of myocardial hypertrophy

- Asymmetric septal hypertrophy
- Basal septal hypertrophy
- Mid-ventricular (with or without apical aneurysm)
- Apical
- Concentric

-Small LV cavity

-Speckled appearance of the myocardium (fundamental imaging – switch off tissue harmonics!)

- -Obstructive (HOCM) / nonobstructive (HCM)
- -Atrial dilatation (especially LA)

3/Specific features

- -SAM (systolic anterior motion) of MV leaflet (HOCM)
 - Mechanism(s)
 - Venturi effect (high outflow velocities in narrowed LVOT)
 - Anterior displacement of MV apparatus
 - Anterior MV leaflet elongated / large area
 - Other SAM provoking conditions
 - MV repair
 - AV replacement for AS
 - Hypovolemia
 - Catecholamines
- -LVOT dynamic obstruction (HOCM)
 - Narrowed LVOT SAM septal contact
 - Increased with certain manoeuvres (standing, exercise)
 - Late-peaking Doppler envelope
 - Mid-systolic AV closure of aortic valve
- -Mid-cavity dynamic obstruction
 - Flow acceleration induced aliasing observed mid-cavitary or toward apex
 - Late-peaking Doppler envelope
 - Mechanism: concentric hypertrophy with hyperdynamic contractility
- -Mitral regurgitation
 - Almost always associated to SAM
 - Eccentric (posterolateral), late-systolic
 - LV diastolic dysfunction
 - Reduced regional myocardial deformation

4/Diagnostic criteria in first relatives

5/Pitfalls

-Differential diagnosis (Chronic hypertension, Amyloid, Pheochromocytoma,

Friedreich's ataxia, Inferior MI with previous LVH, Athlete's heart)

-Dynamic LVOT obstruction not specific

-Apical HCM may be missed – contrast echo may be needed

-IVS measurement including RV papillary muscle, moderator band or trabeculations 6/Treatment guidance

-Medical treatment

-Surgical myectomy (site and extent of resection, immediate results, complications) -Alcohol septal ablation (patient selection, procedure guidance, follow-up)

-AV interval optimization in patients needing DDD for conduction abnormalities

Restrictive cardiomyopathies

1/Classification

-Familial

- Familial unknown gene
- Sarcomeric protein mutations
- Familial amyloidosis
- Haemochromatosis
- Anderson-Fabry disesase
- Glycogen storage disease

-Non-familial

- Amyloid
- Scleroderma
- Endomyocardial fibrosis
- Carcinoid heart disease
- Metastatic cancer
- Radiation induced
- Drugs induced (anthracyclines)
- 2/Typical 2D echo findings
 - -Small to normal LV cavity size
 - -Normal wall thickness
 - -Normal or near-normal LV function
 - -Dilated atria (typical picture: small ventricles with very big atria)
 - -Restrictive filling pattern
 - -Low myocardial early diastolic velocities < 8 cm/s
 - -Regional function inhomogeneity

3/Differential diagnosis: constrictive pericarditis

Arrhythmogenic right ventricular cardiomyopathy (ARVC)

1/RV systolic dysfunction (global or regional) with or without LV dysfunction 2/Histological definition: progressive replacement of myocardium by adipose or fibrous tissue

3/Diagnosis

- -Dilated RV
- -RV aneurysms, outpouchings (at RV inflow, apex, infundibulum)
- -Focal RV wall thinning
- -Abnormal global / regional RV systolic wall motion
- -Abnormal tissue composition on CMR
- -Potential LV involvement

Unclassified cardiomyopathies

1/Takotsubo cardiomyopathy (broken heart)

- -Acute, stress-induced LV dysfunction
- -Mainly women, mostly elderly
- -Findings
 - Hypokinetic apex with hyperkinetic basal segments typical Takotsubo (Apical ballooning, light-bulb like LV)
 - Variant form sparing apex
 - Hypokinesia/akinesia which does not follow a coronary territory of distribution
 - (Normal coronaries)
 - Typical complete recovery in a few weeks
- -Complications
 - LV thrombus
 - Apical rupture
 - RV involvement
 - MR
- 2/Left ventricular non-compaction
 - -Prominent left ventricular trabeculae and deep inter-trabecular recesses
 - -Diagnostic findings
 - Thin compacted epicardial layer and thick non-compacted endocardial layer (Non-compacted: compacted ratio 2:1)
 - Honeycomb appearance in short axis
 - Trabeculae and recesses extend beyond the apex
 - (Usually apical and mid lateral and inferior wall)
 - Colour Doppler flow extends in the recesses
 - -Other findings
 - Global LV systolic dysfunction
 - LV thrombus
 - Abnormal papillary muscle structure
 - -Contrast echo

14. CARDIAC TRANSPLANTATION

1/Morphologic characteristics and function of allograft

- -Reduced septal motion and thickening
- -Exaggerated systolic posterior wall motion and thickening
- -Increased LV mass
- -Biatrial enlargement (donor + recipient)
- -Biatrial anastomoses enhanced echogenecity
 - Suture lines (especially noted in apical 4-chamber view)
 - May be prominent (mass-like appearance)
- 2/RV dimensions
 - -Normal, if pulmonary artery pressures are normal
 - -Dilatation, if pulmonary hypertension preoperatively or perioperatively
- 3/Pericardial effusion
 - -Small effusion common
 - -Large pericardial sac with small effusion
 - -Small heart in large space large pericardium does not follow the myocardium in systole
- 4/Doppler findings in normal allograft
 - -IVRT and mitral PHT may be short immediately after transplant and increase within
 - -6-weeks
 - -Impaired relaxation
 - -Insignificant valve regurgitation

5/Potential complications

-Acute rejection

- Echo findings
 - LV wall thickness increase due to oedema
 - LV systolic function decrease
 - Myocardial echogenecity increase
 - New or increased pericardial effusion
 - Decrease in mitral PHT
 - Shortening of LV IVRT
 - Increase in early diastolic filling velocity (mitral E wave) Increase in E / e'
 - Impaired longitudinal deformation (strain and strain rate) New onset MR
- -Transplant coronary artery vasculopathy
 - Stress echo
 - Coronary flow reserve
- -Pericardial effusion
- -RV systolic dysfunction
- -Injury to tricuspid valve (secondary to repeated RV biopsies)
- 6/Pitfalls
 - -Heart rate and loading conditions changes
 - -Variable timing of recipient and donor atrial contraction
 - -Intersubject and interstudy variation
- 7/Echo guidance of RV biopsies

15. FINDINGS IN SYSTEMIC AND PULMONARY HYPERTENSION

Systemic Hypertension

1/Pathophysiology

-Increased LV afterload => LV hypertrophy + increase in LV mass => diastolic dysfunction

- 2/Findings
 - -Increased LV mass and mass index
 - -LV hypertrophy (develops commencing from the basal part of the IVS)
 - -Increased relative wall thickness
 - -LA dilatation (due to increased LV diastolic pressure)
 - -Aortic root dilatation
 - -MV annulus calcification
 - -Diastolic LV dysfunction
 - -Reduced LV longitudinal function (before the drop in EF)
 - -Global LV systolic dysfunction (hypertensive cardiomyopathy)

3/Role of Echo

-Diagnosis, prognosis, efficacy of medical therapy follow-up

- Regression of hypertrophy
- Improvement of diastolic function / reduction in LV filling pressures
- -Rule out secondary hypertension (coarctation)

4/Complications

- -Aortic regurgitation
- -Aortic dissection

Pulmonary Heart Disease (Cor Pulmonale)

1/Pathophysiology

2/Role of echocardiography

- -Diagnosis of pulmonary hypertension
- -Diagnosis of occult pulmonary hypertension (exercise echo)
- -Quantitation of pulmonary hypertension
- -Diagnosis of pulmonary hypertension consequences
- -Differential diagnosis pulmonary / LA / primary pulmonary hypertension
- -Determine prognosis

3/Differential diagnosis acute / chronic cor pulmonale

-Acute pulmonary hypertension (acute pulmonary emboli)

- Acute RV overload
- Findings
 - RV dilatation and dysfunction, RA dilatation
 - Pulmonary artery dilatation
 - IVS RV pressure overload motion pattern

 $^{\rm o}$ Systolic pulmonary artery pressure (SPAP) can be calculated if there is TR

Reduced pulmonary flow acceleration time

 Thrombus in right heart and /or pulmonary artery (residual or "intransit")

- 60/60 sign, McConnel sign (specific, insensitive)
- Exaggerated MV / TV inflow respiratory variation (similar with tamponade)
- Reduced TAPSE
- High risk indicators
 - RV systolic dysfunction (even if no hypotension)
 - Free-floating right heart thrombus
 - Patent foramen ovale
- -Chronic pulmonary hypertension (chronic cor pulmonale)

Persistent pulmonary hypertension (mean >25mmHg at rest, >30mmHg with exercise)

- Findings
 - RV hypertrophy (differential diagnostic feature), dilation
 - Abnormal RV systolic function
 - IVS RV pressure overload motion pattern
 - Reduced pulmonary flow acceleration time
 - [•] Pulmonary artery can be dilated
 - RA dilatation, interatrial septum (IAS) displaced to the left
 - Pericardial effusion may be present
 - TR allows SPAP calculation (see Chapter 7)

PR – allows mean and diastolic pulmonary artery pressure (DPAP) calculation (see Chapter 7)

Severely elevated SPAP (differential diagnostic feature chronic versus acute)

16. PERICARDIAL DISEASE

Normal Pericardial Anatomy

Pericardial Effusion

1/Diagnosis

2/Differential diagnosis

- -Pericardial effusion / pleural effusion
- -Pericardial effusion / epicardial fat
- -Pericardial effusion / thrombus / haemorrhagic effusion
- 3/Quantitation of pericardial fluid
- 4/Echo indices of tamponade
 - -RA systolic collapse percentage of cardiac cycle length
 - -RV diastolic collapse
 - -Reciprocal changes in ventricular volumes with respiration
 - (Increase RV and decrease LV volume in inspiration and the reverse in expiration)
 - -RV and LV diastolic filling respiratory variation (more than 30%)
 - -Pulmonary and hepatic vein flow changes
 - -IVC plethora
 - -Swinging heart

5/Echo-guided pericardiocentesis (see Chapter 23)

Constrictive Pericarditis

1/Pathophysiology

2/Diagnosis

- -Thickenned pericardium
- -Normal LV size and contractility
- -Dilated atria
- -Posterior wall diastolic motion flattening
- -Septal bounce
 - 2D: IVS displacement towards the LV in inspiration
 - M-mode: IVS abrupt early diastolic posterior motion, flat in mid-diastole,
 - abrupt anterior motion with atrial contraction
- -Dilated IVC and hepatic veins
- -Premature diastolic opening of pulmonary valve

-Doppler

- Prominent "Y" descent on hepatic vein or superior vena cava flow pattern
- Prominent E wave with rapid early diastolic deceleration slope on LV inflow
- LV IVRT increase by >20% in inspiration
- RV IVRT respiratory changes
- Respiratory variation: increase in RV filling and decrease in LV filling
- Respiratory variation of pulmonary venous flow
- Hepatic veins flow reversal mainly in expiration

3/Differential diagnosis from restrictive cardiomyopathy: DTI, Colour M-mode 4/Pitfalls

-COPD

- -Localized constriction (around LV / RV)
- -Irregular cardiac rhythm
- -RV infarction

Pericardial Cysts

1/Diagnosis: round or elliptical echo-free space adjacent to a cardiac chamber

Congenital Absence of Pericardium

1/Total absence

-Unusual echo windows (grossly apparent); shift of heart to left chest

- -Cardiac hypermobility (exaggerated cardiac motion)
- -Apparent RV dilatation in parasternal views (more RV in scan plane)
- -Abnormal IVS motion (mimics RV volume overload)
- -Recommend alternative imaging to confirm diagnosis

2/Partial absence -Potential for herniation / strangulation of part of the heart 3/Associated abnormalities -ASD, bicuspid aortic valve

17. CONGENITAL HEART DISEASE IN THE ADULT

Basic Embryology

1/Primitive cardiac formation 2/Comparison of foetal and postnatal circulation

Segmental Approach

1/Define abdominal situs

-Position of descending aorta with reference to spine

-Presence of absence of inferior vena cava (IVC) and position relative to aorta

- Normal: anterior and rightward
- Absence with dominant azygos vein(s) consider laterality disturbance (isomerism)

2/Cardiac position

-Position (in chest)

-Orientation (position of cardiac apex)

- Levocardia
- Dextrocardia
- Mesocardia

3/Define atrial morphology

-RA characteristics

- RA is defined by a triangular appendage with a wide base
- There are usually inferior and superior caval veins draining to morphological RA but they could be abnormal or absent
- Eustachian valve often seen
- Morphological right atrium (mRA) = an atrium with RA morphology

regardless of which side it is found in

-LA characteristics

- LA is defined by a narrow necked finger like appendage
- Pulmonary veins drain into LA but they could be anomalous or absent
- Morphological left atrium (mLA) = an atrium with left atrial morphology regardless of which side it is found in

-A heart may have one RA and one LA / two mRAs / two mLAs (left atrial isomerism)

-Situs solitus: mLA on left, mRA on right

-Situs inversus thoracalis: mRA on left, mLA on right

-Situs inversus totalis

Situs inversus thoracalis + inverse orientation of the abdominal organs -Left or right atrial isomerism

-Presence of two morphological left or right atria, respectively

4/Define atrioventricular junction

-How many A-V valves?

- Single left sided valve
 - Consider tricuspid atresia
- Single right sided valve
 - Consider mitral atresia
- Common single valve opening to both ventricles
- Consider atrio-ventricular Septal Defect (AVSD)
- Two A-V valves

- TV usually more apically displaced than MV A-V valve offset
- LA to MV to LV and RA to TV to RV A-V concordance
- "Reversed" A-V valve offset (left valve more apical than right AV valve) Consider A-V discordance
- LA to TV to RV and RA to MV to LV A-V discordance
- A-V valve usually concordant with ventricle below TV defines RV below, etc.
- Two AV valve to single ventricle (usually LV)
- Double Inlet Left Ventricle (DILV)

5/Define Ventricular Morphology

- -RV morphology
 - Moderator band
 - Triangular shape
 - Coarse trabeculations
 - TV attachment
 - Tricuspid valve
 - 3 leaflets
 - Less defined papillary muscles (3)
 - Insertion of the septal leaflet to the ventricular septum

-LV morphology

- Elliptical shape
- Smooth, fine trabeculations
- Two papillary muscles
- MV attachment
 - Mitral valve
 - 2 leaflets
 - 2 papillary muscles (antero-lateral and postero-medial)
 - No insertion to the septum

6/Define ventriculo-arterial connections

-Ventriculo-arterial concordance

- The aorta and pulmonary trunk are connected to the LV and RV, respectively
- The aorta is posterior and runs from left to right
- The pulmonary artery is anterior and crosses over the aorta from right to left -Ventriculo-arterial discordance
 - The aorta is connected to mRV and the pulmonary trunk is connected to the mLV $% \mathcal{A}$
 - The great arteries may be parallel and not cross over
- -Single outlet ventricle
 - One great artery atresia (e.g. pulmonary atresia) or
 - Common outlet from both ventricles (common arterial trunk)
- -Double outlet ventricle
 - The two great vessels connected to one ventricle, most commonly the right ventricle as Double Outlet Right Ventricle (DORV).
- 7/Define Great Artery position and course
 - -Normal connection: with normal cross over (see above)
 - -Transposition
 - Describe position of aorta and pulmonary artery relative to each other
 - Aorta may be anterior, left to right or even side by side
 - -Arch of the aorta
 - Right or left sided
 - Define by branching pattern: innominate artery to right or left

8/Bubble-Contrast echocardiography for further differentiation

- -Agitated saline solution
- -Right-to-left shunt
- -Systemic venous return anomalies
- -Pulmonary arterio-venous fistula

Shunt lesions

1/Atrial septal defect

- -Types (Secundum, Primum, Sinus venosus, Coronary sinus)
- -Single or multiple defects
- -Associated abnormalities
- -Pathophysiology, hemodynamics, Eisenmenger syndrome
- -2D echo findings
 - Signs of RV volume load +/- pressure overload
 - RV, RA enlargement
 - RV systolic function may be impaired
 - Septal defect: subcostal, modified P-LAX, P-SAX, 4ch (possible drop-out artefact)

• Size, location and septal rims best described by TOE (for percutaneous closure)

- Associated A-V valve abnormalities (ASD primum as part of AVSD spectrum)
- Associated partial anomalous venous drainage especially in sinus venosus ASD

-Doppler findings

- Low velocity ASD
 - Flow almost throughout the cardiac cycle
 - Starts in early systole and has a broad peak in late systole and early diastole
- Increased TV inflow compared with MV inflow
- Pulmonary artery pressure could be elevated (estimated from TR + RAP)
- Qp/Qs (significant if >1.5)
- Colour flow mapping (direction of the shunt)
- Bubble contrast (small shunt, or patent foramen ovale)

-Indications for TOE

- Peri-ASD rim assessment
- Percutaneous closure guidance
- Signs of volume overload raising suspicion of left to right shunt
- Suspected superior or inferior sinus venosus ASD or coronary sinus ASD
- Suspected partial anomalous pulmonary venous drainage

-Additional information with 3D echo

- Findings after atrial septal defect repair
- Residual shunt
- Persistent pulmonary hypertension
- Persistent RV dilatation and/or dysfunction
- Deformation of IAS by device
- Patch of ASD closure more likely seen post ostium primum closure
- Abnormal anterior left AV valve leaflet appearance post ostium primum+ cleft closure

2/Ventricular septal defect

-Define septal site: inlet, perimembranous, apical, outlet

- -Single or multiple defects
- -Physiology, hemodynamics, Eisenmenger syndrome
- -Associated lesions
 - Valvular regurgitation (e.g. AR in sub-aortic defect)
 - Sub-aortic membrane
 - Double chamber RV

-Echo findings

- Type, location and size of defect: P-LAX, P-SAX, 4Ch
- RV and LV size and function (increased with large shunt)
- LA size (increased with large shunt)
- Signs of spontaneous closure
 - Aneurysm
 - TV attachments in perimembranous VSD
- RV outflow tract
- -Doppler findings
 - Interventricular gradient
 - Qp/Qs
 - SPAP rise
 - Colour flow mapping (shunt flow, other abnormalities)
 - AR

-Findings after ventricular septal defect repair

- Residual shunt
- Persistent pulmonary hypertension
- AR, TR, MR
- Closure device
- Patch of closure

3/Atrio-ventricular septal defects

-Types

- Partial (no VSD)
- Complete (atrial and ventricular components)

-Associated lesions

- Abnormal A-V valves
- -Possible LV outflow tract obstruction (LVOTO)
- -Echo findings
 - 4ch: ASD +/- VSD
 - Valvular lesion
 - No A-V valve offset
 - May have common A-V valve or separate A-V valve orifices
 - A-V valve regurgitation common
- -Findings post surgery

Residual valvar lesion (left or right A-V valve – stenosis / regurgitation)
 4/Patent ductus arteriosus

-Physiology, hemodynamics

- -Echo findings
 - LV volume overload due to left to right shunt
 - Dilated LA
 - Enlarged pulmonary artery
- -Doppler findings
 - Specific views (high short axis, suprasternal, high parasternal)
 - SPAP (functional significance)
 - Ductal flow profile changes with SPAP rise (usually continuous flow with systolic peak)
 - Qp/Qs
- -Associated lesions (patent ductus arteriosus is usually an isolated lesion) -Findings after patent ductus arteriosus repair
 - Post coils closure flow acceleration +/- gradient in the descending aorta
 - Residual shunt
 - Peripheral pulmonary artery stenosis
 - Abnormal proximal descending aorta flow profile if duct device protrudes into aorta

5/Partial anomalous pulmonary venous drainage

-Physiology, hemodynamics, common connections

-Associated lesions (mainly associated with sinus venousus ASD)

6/Total Anomalous Pulmonary Venous Drainage

-Uncommon in adult CHD, usually presents in infancy

-May be associated with laterality disturbance (isomerism)

Left ventricular outflow obstruction

1/Isolated

-Congenital valvular AS (see chapter 12 for aetiology and echo assessment)

-Post repair restenosis + / - regurgitation

-Subvalvular AS

-Regrowth after resection

-Supravalvular AS

Associated with Williams syndrome

2/Associated with other left heart obstruction

-Supramitral membrane

-Coarctation

Coarctation of the aorta

1/Frequently associated with bicuspid aortic valve and ascending aortic aneurysm 2/Suprasternal view for anatomy and presence of turbulent flow

3/Doppler

-Measure V_{max}

-With coexistent AS, the gradient can overestimate severity (high V1)

-Assess diastolic flow pattern

4/Assess LV dimensions, LV function and hypertrophy

5/Post-repair

-Recoarctation

-Aneurysm of repair site (MRI assessment better)

RV outflow obstruction

1/Types

-Valvular - pulmonary stenosis

-Subvalvular (infundibular)

-Supravalvular

2/Associated abnormalities

-Tetralogy of Fallot

-Noonan syndrome

-VSD

-Peripheral pulmonary artery stenosis (right or left main pulmonary artery) 3/Echo findings

-Identify level(s) of obstruction

-Quantify severity of obstruction

-Identify associated abnormalities

-Always check RVOT velocity

4/Findings postintervention (percutaneous repair or surgery)

-Residual or recurrent obstruction

-Pulmonary regurgitation

-Deterioration of RV function

Disease of the mitral valve

1/MS

- -Parachute MV
- -Double orifice MV
- -Post cleft repair

-Either true Cleft MV or as part of AVSD

2/MR

-Define direction of MR flow

-Post AVSD (best view P-SAX at the level of AV valve)

3/Cor triatriatum

-Membrane above LA appendage

-Pre-mitral valve flow acceleration / LV inflow gradient

Disease of the TV

1/Ebstein's Anomaly

-Echo findings

- Apical displacement of septal +/- posterior TV leaflets into the right ventricle
- Resultant partial "atrialisation" of RV inflow
- Large RA with consequently small remaining functional RV

• TR

- RV volume overload
- Degree of RV +/- LV dysfunction
- Associated findings

+/- ASD

- +/- RV outflow tract obstruction
- 3D echo very useful
- -Role of Echo in patient selection for surgery
 - Severity assessment
 - Feasibility of surgical repair assessment (leaflets mobility, insertion and length)
 - ASD assessment

-Post-repair assessment: tricuspid regurgitation / stenosis, RV / LV function

Complex congenital lesion

1/Tetralogy of Fallot after Repair

-Pathophysiology - 4 coexistent abnormalities

VSD

- RV hypertrophy
- RVOT obstruction
- Overriding aorta
- -Repair procedure

-Post-surgical assessment

- Residual RVOT obstruction
- Degree of PR
- RVOT aneurysm
- RV dimensions (dilatation) and function (role of 3D echo)
- TV annulus dilatation, TR

• PR

- Pulmonary trunk, left and right pulmonary arteries dimensions
- Pulmonary homograft assessment (if implanted)
- AR and aortic root dilatation
- Residual VSD
- LV function

2/Transposition of the great arteries

- -Pathophysiology
- -Associated findings
- -Repair procedures
 - Atrial switch (Mustard Senning)
 - Arterial switch (normal physiology)
- -Post-surgical assessment
 - Atrial switch
 - Morphological RV is the systemic ventricule
 - RV function assessment
 - Septal shift
 - TR (systemic atrio-ventricular valve)

 Baffle assessment (stenosis / regurgitation of pulmonary / caval venous baffle)

- Arterial switch
 - No-aortic valve dysfunction, neoAR most common Dilatation of aortic root
 - Branch PA stenosis (S/P Le Compte manoeuvre)

Congenitally corrected transposition of the great arteries

- 1/Pathophysiology
- 2/Associated findings
- 3/Echo assessment
 - -RV systolic function assessment (systemic ventricle)
 - -TR assessment (systemic atrioventricular valve)

Univentricular heart

- 1/Pathophysiology
- 2/Palliation procedures
 - -Superior cavo-pulmonary artery connection (e.g. Bidirectional Glenn shunt) -Fontan repair: inferior cavo-pulmonary artery connection (various surgical techniques)
- 3/Echo assessment
 - -Assess pathway patency, spontaneous echo contrast
 - -Shunt colour Doppler flow assessment (turbulence)
 - -Surgical fenestration from tunnel to common atrium
 - -Single ventricular function assessment
 - -Single atrio-ventricular valve regurgitation assessment

Other

1/Persistent left superior vena cava

- -Echo findings
 - Marked coronary sinus dilatation, best seen in parasternal long axis
 - Suprasternal view shows left superior vena cava
 - Agitated saline injected in the left arm
 - Appears first in the coronary sinus and then in the right atrium

18. CARDIAC MASSES AND POTENTIAL SOURCES OF EMBOLI

Vegetations

1/Major risk sources

2/Infective endocarditis - embolic potential increases with vegetation size and mobility 3/Nonbacterial thrombotic endocarditis

Giant Lambl's excrescences

1/Minor / unclear sources of emboli

Thrombi

1/LV thrombus

-Predisposing features

• Regional wall motion abnormality (acute myocardial infarction, apical akinesia)

- LV aneurysm / pseudoaneurysm
- Global LV systolic dysfunction
- Non-compaction
- -Association with spontaneous echo contrast

-Location

- Adjacent to akinetic or hypokinetic LV segment
- -Diagnostic features
 - Echo dense mass with defined margins, distinct from the endocardium
 - Seen throughout systole and diastole in at least two views
 - Echogeneity differs from myocardium
 - Low echogeneity new thrombus
 - High echogeneity, brightness old thrombus

-Types of thrombus

- Mural (flat surface) old, organised, layered, laminated
- Protruding (convex surface) fresh, non-organised
- -Embolic potential
- Depends on size, mobility, protrusion into cavity, age of thrombus -Scanning tips

• Use high-frequency for better resolution, especially to investigate the cardiac apex

- Decrease depth
- Position focus near apex / near investigated area
- Use low transmit power and gain
- Add modified / nonstandard views
- TTE makes diagnosis
- Use low flow (low aliasing velocity) colour Doppler assessment
- Use contrast when necessary

-Pitfalls

- Near-field and ring-down artifact (for apical thrombus)
- Prominent trabeculations
- Papillary muscles
- False tendons
- Organised flat thrombi more difficult to diagnose

2/LA thrombus

-Predisposing features

- Atrial fibrillation
- MS
- Prosthetic mitral valve
- LA enlargement

- Low cardiac output
- IAS aneurysm
- -Association with spontaneous echo contrast
- -Location

• LA

- LA appendage (poorly imaged with TTE)
- Thrombus straddling through a patent formen ovale (PFO)

-Diagnostic features

- Echo dense mass with defined margins, distinct from the endocardium
- Seen in at least two views
- Echogeneity differs from surrounding tissues
 - Low echogeneity new thrombus
 - High echogeneity, brightness old thrombus
- -Differential diagnosis
 - Warfarin ridge (TOE)
 - Pectinate LAA muscle (TOE)
 - MV prosthetic valve shadow artefact
- -Scanning tips
- TOE needed to exclude LA thrombus (more sensitive than TTE) -Embolic potential
 - Depends on size, mobility, protrusion into cavity, age of thrombus
 - Contraindicates
 - Electric cardioversion of atrial fibrillation
 - Atrial fibrillation ablation
 - MV clip
 - LAA device closure
 - Balloon mitral valvotomy (relative contraindication)

3/RA thrombi

- -Predisposing features
 - Atrial fibrillation
 - RA enlargement
 - Catheters, devices leads
 - IAS aneurysm
 - Deep vein thrombosis
- -Clinical significance
 - Embolization (pulmonary / paradoxical)
 - Intermittent RV outflow obstruction with syncope / "obstructive shock"
 - Thrombi on catheters and devices leads can become infected
- -In transit (venous cast)
- -Differential diagnosis
 - Eustachian valve, Crista Terminalis, Chiari network, Thebesian valve
 - Reverberation artifacts
 - Lipomatous IAS infiltration
- 4/RV thrombi
 - -Rare
 - -In transit (venous cast) imminent pulmonary emboli
 - -Predisposing features
 - ARVC
 - Devices leads
 - -Differential diagnosis
 - Moderator band

Cardiac Tumors

1/Primary

-Benign

- Myxoma
- Papillary fibroelastoma
- Lipoma
- Fibroma
- Hemangioma
- Miscellaneous others
- -Malignant
 - Sarcomas
 - Angiosarcoma Rhabdomyosarcoma Fibrosarcoma Extraskeletal osteosarcoma
 - Mesothelioma
 - Malignant lymphoma
 - Miscellaneous others
- 2/Secondary (metastatic)
 - -Metastasis to visceral pericardium
 - -Tumors invading right heart via IVC
 - Renal cell carcinoma (hypernephroma)
 - Hepatocellular
 - Uterine tumors
 - -Tumors invading left heart via plmonary veins
- 3/Role of echocardiography in diagnosis
 - -Detection and characterization
 - Morphology
 - Location, single, multiple
 - Site and nature of attachment
 - Infiltration (suggests malignant)
 - Contrast study to assess vascularity
 - -Differential diagnosis
 - Normal variants
 - Thrombus
 - Trabeculae / non-compaction
 - Iatrogenic material
 - Intracardiac devices
 - Preserved segments of subvalvular apparatus in MV surgery
 - -Guidance of biopsy, surgery

Miscellaneous Non-Neoplastic Intracardiac Masses

1/Mitral annulus calcification (MAC)

- -Diagnosis
 - Bright, increased echodensity in region of annulus
 - May extend into leaflets
 - May infiltrate myocardium
 - May obscure visualization behind it
 - May be associated with thrombus
- -Consequences
 - Mitral regurgitation
 - Mitral stenosis
 - Mainly when extending into both leaflets
 - Usually mild

Embolisation

2/"Atypical" mitral annulus calcification

- -Necrotic liquefaction of MAC, sterile, caseous mitral annular "abscess"
- -Creates suspicious-appearing mass on CXR and echo
 - Differential diagnosis: Tumor, infective endocarditis (abscess)
 - Cardiac surgery may be performed unnecessarily
- -Echo features
 - Localized mass (rather than "ring-like")
 - Usually beneath posterior leaflet
 - Often outer echo-dense rim with central lucency

3/Cystic masses

-Blood cyst

- Congenital
- Small, round, single or polilobate
- Attached mainly to A-V valves (closure lines, chords, papillary muscles)
 Echinococcal cyst
 - Septated cysts, intramyocardial / protruding in cardiac chamber
 - LV, IVS

Extracardiac "Masses"

1/Cysts

-Pericardial

-Bronchogenic

2/Mediastinal tumors

3/Aorta

-Tortuous

-Aneurysms

4/Hiatus hernia

Structures Mistaken for Abnormal Cardiac Mass

1/Left atrium

-Ridge between left superior pulmonary vein and LA appendage (warfarin ridge)

-Atrial suture line after cardiac transplant

- -Inverted left atrial appendage
- -IAS aneurysm

-Lipomatous IAS infiltration

-Pectinate muscles in LA appendage

-Tortuous descending thoracic aorta "compressing" LA

-LV lead in coronary sinus

-ASD / PFO / LAA closure devices

2/Right atrium

-Crista terminalis

-Eustachian valve

-Chiari network

-Lipomatous IAS infiltration

-RA appendage trabeculations

- -Atrial suture line after cardiac transplant
- -Catheters, central venous lines, pacemaker wires, devices leads

-ASD / PFO closure devices

3/Left ventricle

- -Papillary muscles
- -False tendons
- -Prominent muscular trabeculations
- -Prominent MAC

4/Right ventricle

- -Moderator band
- -Papillary muscles
- -Catheter or pacemaker wire

5/Aortic valve

- -Nodules of Arantius
- -Lambl's excrescences
- -Aortic cusp imaged *en face* in diastole (TEE)

6/Mitral valve

- -Redundant chordae
- -Myxomatous mitral valve tissue

7/Pericardium

- -Epicardial adipose tissue
- -Fibrinous debris in chronic pericardial effusion

19. DISEASES OF THE AORTA

Anatomy of the aorta

1/Ascending aorta

-Aortic root: annulus, sinuses of Valsalva, sinotubular junction

-Tubular ascending aorta

2/Aortic arch

3/Descending aorta

Echo assessment

1/TTE

-Parasternal long axis view (aortic root, part of the tubular ascending aorta) -Suprasternal view (aortic arch, part of the ascending aorta and part of the descending aorta)

2/TOE

-Aortic root and tubular ascending aorta – anterior upper oesophageal views

- Short axis (30°-70°)
 Lang axis (1200, 1600)
- Long axis (120°-160°)

-Descending aorta (posterior lower and upper oesophageal views)

- Short axis (0°)
- Long axis (90°)
- -Aortic arch (anterior upper oesophageal views)
- -Blind spot, crossing airway

Aortic Dissection

1/Classification

-Acute / chronic

-Class I, II, III, IV, V (see ESC Recommendations)

-Types I, II and III (DeBakey) or types A and B (Stanford)

2/Echo findings

- -Dissection flap
- -Dilated aorta
- -Thickened aortic wall (intramural hematoma)
- -Aortic regurgitation (assess severity and mechanism)

-Pericardial and / or pleural effusion

-Compression of left atrium

-Entry site / re-entry site (identification and differential diagnosis)

-True lumen / false lumen (differential diagnosis)

-Coronary artery involvement (direct flap visualisation / regional wall motion abnormalities)

-Visualised major branch involvement

-Aortic rupture

-False lumen thrombosis

3/Transesophageal echocardiography

-Sensitivity / specificity

-Advantages / superiority over TTE / comparison with CT and MRI

-Disadvantages / not first line aortic dissection assessment, only in theatre preoperatively

4/Pitfalls

-Reverberations, catheters

-Mirror-image artefacts

-Spiral flow down descending aorta

-Thoracic aortic aneurysm with mural thrombus

-"Blind spot" (can miss type II dissection)

5/Post repair findings

-Aortic valve repair or replacement, aortic regurgitation

-Pericardial effusion

-False lumen persistence, assessment of flow in true / false lumen

Intramural hematoma ("atypical" aortic dissection)

1/Pathogenesis

- -Small intimal tears
- -Ruptured vasa vasorum
- -Penetrating ulcer

-Trauma

2/Echo findings

-Excentric or circumferential thickening of aortic wall

-Absence of dissection flap

-Preserved aortic lumen

3/Differential diagnosis

-Aortic aneurysm with mural thrombus

-Atherosclerotic thickening of aortic wall

Thoracic Aortic Aneurysm

1/Definition

-Dilatation of the aorta which contains all 3 layers (intima, media, adventitia) 2/Pathogenesis and aetiology

-Atherosclerosis

-Medial degeneration

-Idiopathic (annuloaortic ectasia)

-Marfan syndrome

-Other inherited disorders

-Associated with bicuspid aortic valve

-Association with aortic coarctation

-Aortic dissection with dilatation of persisting false lumen

-Trauma with incomplete aortic rupture

-Syphilis

-Mycotic (bacterial, fungal, tuberculous aortitis)

-Non infectious aortitis (giant-cell, Takayasu's syndrome)

3/Location

Ascending aorta / aortic arch / descending aorta / thoraco-abdominal aorta

4/Dimensions

-Measured on 2D in end-diastolic frame (maximum dimensions are measured in systole)

-Absolute values / Notmograms / Indexed measurements per body surface area 5/Echo findings

6/Role of TOE

7/Differential diagnosis: aortic dissection with thrombosed false lumen

8/Indications for surgery

Traumatic aortic lesions

1/Types of trauma

-Blunt chest trauma

- Consequences
 - Contusion
 - Aortic rupture

Complete transection – may be contained by surrounding tissues Pseudoaneurysm

- Aortic dissection
- Intramural haematoma
- Laceration of the aortic wall
 - Usually horizontal
 - Small (limited) / Large (circumferential)
 - Extend outward from the intima
- Location

Usually just distal to left subclavian artery origin at ligamentum arteriosum

Frequently proximal thoracic aorta

- -Iatrogenic trauma
 - Consequence
 - Aortic dissection
 - Usually retrograde when catheter induced
 - Dislodgement of debris from protruding or mobile atheromas
 - Circumstances
 - Heart catheterisation
 - Angioplasty of aortic coarctation
 - Cardiac surgery crossclamping of the aorta
 - Intra-aortic balloon pump

2/Echo findings

-See aortic dissection and intramural haematoma

3/TOE advantages and limitations

Aortic Atherosclerosis

1/TOE findings

-Atheroma (size / location / mobility component / ulceration / thrombus) -Grading

- I no or minimal intimal thickening
- II intimal thickening 1-3.9 mm without atheroma
- III atheroma < 4 mm

IV – intimal thickening or atheroma > 4 mm

V – ulcerated or mobile atheroma

- 2/Clinical relevance
 - -Risk of embolisation
- 3/Epiaortic ultrasound imaging

-Aortic clamping site selection in the operating room

Sinuses of Valsalva pathology

1/Aneurysm

- -Echo findings
 - Dilated sinus of Valsalva round or fingerlike (windsock) aneurysm
 - Size / size variation during cardiac cycle
- -Complication
 - Rupture

2/Rupture

-Aetiology

- Aneurysm
- Aortic root abscess
- Iatrogenic (during procedures, e.g. ablation or aortic valve replacement)
 -Consequences
 - Right coronary cusp rupture communication with RV or RA
 - Non coronary cusp rupture communication with LA
- -Echo findings
 - Colour Doppler flow through pathological communication
 - Continuous flow / high velocity flow
 - RV dilatation / volume overload

20. STRESS ECHOCARDIOGRAPHY

Basic Principles

1/Determinants of myocardial oxygen demand

2/Ischaemic cascade (sequence of events in ischemia)

3/Regional myocardial function in ischaemia / myocardial infarction

4/Stunning / hibernation

5/Coronary flow reserve

6/Coronary territories of distribution

7/Principles of valves assessment with stress

8/Haemodynamic changes with exercise

Procedural considerations

1/Echo considerations

-Imaging

- 2D / 3D
- Contrast-enhanced echocardiography
- DTI and speckle tracking echocardiography in stress echo
- -Views for evaluation of regional LV wall motion
- -Digital acquistion protocols

-Parameters assessed for evaluation of valves disease and haemodynamics

2/Indications

- -Assessment of existence of inducible ischaemia
- -Assessment of existence of viability
- -Assessment of valve disease
 - AS
 - MS
 - MR

-Assessment of haemodynamics

- HOCM exertion induced increase in obstruction
- Pulmonary hypertension increase in SPAP

3/Types of stress

- -Exercise
 - Treadmill
 - Bicycle: upright / supine
- -Pharmacologic
 - Dobutamine
 - + / atropine
 - + / pacing
 - Adenosine
 - Dipyridamole

4/Pre test preparation

- -Stop beta-blockers, ivabradine, etc for dobutamine test
- -Stop aminophylinne before adenosine or dipyridamole test
- -Stop of xanthines containing drinks before adenosine or dipyridamole test 5/Contraindications
- -Relative
 - -Relative
- 6/Safety / complications

7/Patient monitoring requirements / resuscitation facilities / drugs to treat complications 8/Reasons for termination

- -Test end
 - Completion of protocol
 - Achievement of target heart rate / workload
 - -Positive test
 - Induced regional wall motion abnormalities
 - Severe chest pain
 - -Side effects
 - Systemic hypertension
 - Systolic blood pressure >220mHg
 - Diastolic blood pressure >120mmHg
 - Symptomatic hypotension / vaso-vagal episode
 - Tachyarrhythmias
 - Patient request
- 9/Interpretation
 - -Test positive for ischaemia
 - Induced regional wall motion abnormality
 - Reduced coronary flow reserve
 - -Test positive for viability
 - Continuous improvement / bi-phasic response
 - Inotropic reserve / contractile reserve
 - Wall motion score index

10/Limitations

Exercise Echo

- 1/Basic principles
- 2/Exercise protocols
 - -Treadmill / Bicycle
 - -Depending on indication
- 3/Peak / Post-peak image acquisition

4/Contraindications

Dobutamine Stress Echo

1/Basic principles 2/Infusion protocol 3/Role of atropine 4/Contraindications

-Severe systemic hypertension on presentation

-Tachyarrhythmia on presentation

5/Side effects

- -Hypotension
- -LV outflow tract obstruction

Adenosine Stress echo

1/Basic principles 2/Infusion protocol 3/Contraindications -Asthma -Bradycarrhythmia

Dypiridamole Stress Echo

1/Basic principles 2/Infusion protocol 3/Contraindications -Asthma -Bradyarrhythmia 4/Response -Hyperdynamic response → microvascular disease 5/Side effects -Hypotension

21. TRANSOESOPHAGEAL ECHOCARDIOGRAPHY

Procedural considerations

- 1/Laboratory requirements
 - -Equipment
 - Echo equipment
 - Monitoring equipment
 - Suction
 - Resusciation equipment
 - -Consumables
 - Bite-guards
 - Cover sheaths
 - Disinfectant wipes
 - Three-way taps and luer locl syringes for agitated saline test

-Drugs

- Local anaesthetic
- Sedative / sedative-antidote (e.g. midazolam / flumazenil)
- -Oxygen saturation, pulse and blood pressure monitoring

-TOE probe disinfection and storage

2/Patient preparation

- -History and physical examination
- -Gain i.v. access
- -Remove mobile denture
- -Nasal oxygen cannula if sedation is used
- -Remove naso-gastric tube in intubated patient
- -Deflate the endotracheal cuff in intubated patient if necessary
- -Local anaesthesia
- -Conscious sedation if needed

3/Patient recovery

4/Technique

- -Probe insertion
- -Probe advancement / withdrawal / manipulation
- -Probe position
 - Upper / Lower oesophageal
 - Transgastric
- -Standard and additional views relative to probe position
- -Structures visualised relative to probe position / angle
- -Bi-plane / multiplane / 3D examination
- 5/Indications
 - -Assessment for cardiac source of emboli
 - -Infective endocarditis
 - -Assessment of MR
 - -Assessment of prosthetic valves
 - -Aortic dissection (not first line test)
 - -Intervention guiding see Chapter 23
- 6/Contraindications
 - -Absolute
 - Perforated visous
 - Oesophageal pathology (stricture, trauma, tumor, sceroderma, Mallory-Weiss tear, varices, diverticulum)
 - Active upper gastro-intestinal bleed
 - Recent upper gastro-intestinal surgery
 - Oesophagectomy, oesphago-gastrectomy
 - Respiratory failure if the patient is not intubated

-Relative

- Atlantoaxial joint disease
- Severe cervical arthritis causing restricted cervical mobility
- History of chest radiation
- Symptomatic hiatus haernia
- History of gastro-intestinal surgery
- Recent upper gastro-intestinal bleed
- Oesophagitis, peptic ulcer
- Barrett's oesophagus
- Dysphagia
- Thoraco-abdominal aneurysm
- Coagulopathy, thrombocytopaenia
- Oropharyngeal distortion
- Unwilling or uncooperative patient
- 7/Complications
 - -Arrhythmias
 - -Transient hypotension or hypertension without or with complications
 - -Laryngospasm, bronchospasm
 - -Respiratory failure
 - -Aspiration
 - -Oropharyngeal, oesophageal or gastric laceration / perforation
 - -Bleeding
- 8/Diagnostic value
- 9/Limitations

22. ECHO FINDINGS IN ACUTE AND CHRONIC CLINICAL SCENARIOS

Athlete's Heart

1/Morphologic changes related to type, intensity, and duration of exercise

-Dynamic (isotonic) exercise (running, ski, soccer)

- Physiopathology
 - Predominantly volume load
- Echo findings
 - Increased LV diastolic dimensions
 - Increased LV wall thickness, mass ("eccentric" LV hypertrophy)
 Wall thickness gender / race / age variation
 - Increased RV diastolic dimensions
 - Increased IVC dimensions but with normal respiratory variation
- -Static (isometric) exercise (weight lifting, gymnastics, wrestling)
 - Physiopathology
 - Predominantly pressure load
 - Echo findings
 - No significant increase in heart cavity size
 - Predominant increase in wall thickness ("concentric" LV hypertrophy)

2/Normal diastolic parameters

3/Normal regional function (DTI and deformation imaging)

4/Reversibility with deconditioning

5/Could have increased left atrial volume index

6/Differential diagnosis:

-Hypertrophic cardiomyopathy

-Dilated cardiomyopathy

Heart during pregnancy

1/Physiological changes

-Increased blood volume + decreased systemic vascular resistance => increased SV and CO

-Maximum volume overload: third trimester and immediately postpartum

2/Echo findings

-Dilation of cardiac chambers (LV, RV, LA, RA)

-Increased LV SV

-Change in MV / TV leaflet coaptation +/- consequent mild MR / TR

- -Increased TR velocity
- -Small pericardial effusion
- -Type I filling pattern near term
- -Reversible changes

3/Peripartum cardiomyopathy

Systemic Diseases

1/Carcinoid

-Potential echo findings

- Right heart involvement predominant
 - TV leaflets / PV cusps thickened, retracted with restricted motion
 - RV and RA enlargement
- Left heart involvement less common
 - [•] In patients with intracardiac shunt (PFO) or bronchial carcinoid
 - MV leaflets / AV cusps thickened
 - Moderate to severe MR
- Small pericardial effusions
- -Differential diagnosis
 - Drug induced valvular disease
 - Libman Sacks

2/Haemochromatosis

-Potential echo findings

- Ventricular wall thickness usually normal
- Diastolic dysfunction in early stage
- Dilated cardiomyopathy (common)
- Restrictive cardiomyopathy (uncommon)
- Mild valvular regurgitation

3/Sarcoid

-Potential echo findings

Regional wall motion abnormalities not following coronary territory of distribution

- Focal septal thinning
- Basal posterolateral wall thinning
- Other
- Diastolic dysfunction preceding systolic dysfunction
- Cor pulmonale
 - Secondary to lung sarcoid disease
 - Pulmonary hypertension, RV and RA enlargement
- Papillary muscle dysfunction resulting in MR
- Pericardial effusion (uncommon)
- Restrictive cardiomyopathy in case of extensive myocardial infiltration
- Dilated cardiomyopathy (4-chamber enlargement)

4/Amyloid

-Potential echo findings

Increased LV / RV wall thickness

 Increased myocardial echogenicity ("granular sparkling" – take off harmonics)

- Restrictive cardiomyopathy
 - Systolic function early preserved / late reduced
 - Diastolic dysfunction
 - Bi-atrial dilatation
- Valvular thickening and regurgitation (usually mild)
- Could have SAM
- Atrial thrombus
- Pericardial effusion
- 5/Connective tissue diseases
 - -Rheumatoid arthritis
 - Potential echo findings
 - Pericardial
 - Effusion (acute pericarditis)
 - Constrictive pericarditis
 - Effusion-constrictive

Myocardial

Systolic dysfunction (myocarditis, nodules, accelerated atherosclerosis)

- RWMA related with coronary arteritis rare
- Diastolic dysfunction
- Secondary amyloidosis
- Valvular / endocardial

Valve thickening and regurgitation (valvulitis)

- Nodules
- AR, aortic dilatation
- Aortic aneurysm, wall thickening aortitis (rare)
- Secondary pulmonary hypertension (rare)

-Systemic Lupus Erythematosus

- Potential echo findings
 - Pericardial

Effusion (often clinically silent)

Cardiac tamponade uncommon

- Myocardial
 - Global LV dysfunction
 - RWMA (accelerated atherosclerosis, coronary vasculitis, emboli)
- Valvular / endocardial
 - Thickening (especially mitral, aortic)
 - Nodules
 - Regurgitation
 - Nonbacterial vegetations (Libman-Sacks)
 - Usually <1 cm₂
 - Irregular borders
 - No independent motion
 - MV prolapse
- -Antiphospholipid Syndrome
 - Potential echo findings
 - Intracardiac / aortic thrombi
 - LV systolic dysfunction
 - RWMA (emboli)
 - Dilated cardiomyopathy
 - Valvular involvement primarily regurgitation
 - Pulmonary hypertension
- -Scleroderma
 - Potential echo features
 - Pericardial
 - Effusion / tamponade / constriction
 - CREST syndrome: symptomatic pericarditis
 - Myocardial
 - LV hypertrophy with systemic hypertension
 - LV dilatation
 - LV systolic dysfunction
 - Cardiomyopathy (dilated or restrictive)
 - Pulmonary hypertension
 - RV hypertrophy
 - Valvular involvement thickening
- -Mixed Connective Tissue Disease
 - Cardiac involvement
 - Pericarditis
 - RWMA due to coronary arteritis (rare)
 - Global / regional systolic dysfunction due to myocarditis (rare)
 - Pulmonary hypertension
 - MV prolapsed
- 6/Ankylosing spondylitis

Potential echo findings

- Dilatation of aortic annulus / sinuses of Valsalva
- Aortic valve thickening
- AR due to cusps thickening / root dilatation
- Thickening of aorto-mitral junction ("subaortic bump")
- MV prolapse
- LV systolic dysfunction
- Pericarditis / pericardial effusion (rare)

7/Reiter's syndrome

-Potential echo findings (see ankylosing spondylitis)

- 8/Marfan Syndrome
- -Echo findings
 - See chapter 19
 - Aortic root dilatation
 - Annulus and sinuses of Valsalva
 - Annulo-aortic ectasia
 - Ascending aorta dilatation Fusiform aneurysm of the ascending aorta
 - AR due to aortic root dilatation
 - Aortic dissection
 - Mitral valve myxomatous / prolapse / dilated annulus / MR
 - LVSD
- 9/Giant cell arteritis
 - -Potential echo findings
 - Aortic aneurysm / dissection
 - Aortic root dilatation / thickening of aortic valve cusps
 - LV / RV systolic dysfunction due to myocarditis
 - Pericardial effusion (pericarditis)
- 10/Takayasu arteritis
 - -Potential echo findings
 - Aortic dilatation / aortic wall thickening (seen mainly on TOE)
 - AR
 - Stenosis / occlusion of large vessels e.g. subclavian artery
- 11/Kawasaki Disease
 - -Potential echo findings
 - Coronary artery aneurysms (small <4 mm, medium 4-8 mm, giant >8 mm)
 - Pericardial effusion (pericarditis)
 - RWMA due to coronary insufficiency or myocarditis
 - Secondary mitral regurgitation
- 12/Syphilitic aortitis
 - -Potential echo findings
 - Aortic root dilatation / aneurysm
 - AR
 - Aortic dissection
 - Aortopulmonary fistula
- 13/Hypereosinophilic syndrome (Löffler's)
 - -Potential echo findings
 - LV / RV apical cavity obliteration
 - LV / RV thrombus
 - Despite normal regional wall motion (due to endocardial inflamation)
 - Cardiomyopathy
 - Restrictive
 - Dilated (myocarditis)
 - MR (often moderate to severe) / TR of variable severity
 - Pericardial effusion
- 14/Churg-Strauss syndrome
 - -Potential echo findings
 - Pericardial effusion
 - Restrictive cardiomyopathy due to endomyocardial fibrosis
 - MR / TR due to papillary muscle involvement in endomyocardial fibrosis Increased echogeneity of the myocardium not due to scar of infarction

15/Wegener's granulomatosis

-Potential echo findings

- Pericardial effusion
- 16/Endocrine diseases
 - -Hyperthyroidism
 - Hyperdynamic LV
 - Caution needed in the interpretation of Doppler velocities
 - LV systolic dysfunction (usually tachycardia-induced)
 - -Hypothyroidism
 - Pericardial effusion
 - Dilated cardiomyopathy
 - -Pheochromocytoma
 - LV systolic dysfunction
 - (Catecholamine-induced / tachycardia induced / due to hypertension)
 - LV dilatation
 - LV hypertrophy
 - Dynamic LVOT obstruction could be present
 - -Acromegaly
 - LV hypertrophy concentric or eccentric
- 17/HIV disease (AIDS)
 - -Potential echo findings
 - Pericardial effusion / thickening / constriction
 - Dilated cardiomyopathy myocarditis / neoplastic infiltration
 - Cardiac masses tumors / vegetations
 - Pulmonary hypertension
- 18/Systemic infection/sepsis
 - -Potential echo findings
 - Reversible LV dilatation / systolic dysfunction (myocardial depression)
 - Pericardial and pleural effusions
- 19/Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu)
 - -Hyperdynamic circulation
 - -Echo findings
 - LV dilated
 - Bubbles study -> late positive in pulmonary arterio-venous malformations
- 20/Chagas disease
 - -Echo findings
 - Acute
 - LV dilatation, reduced systolic function, RWMA
 - Chronic
 - LV apical aneurysm with thrombus
 - LV posterior wall hypokinesia with minimal IVS involvement
 - RV systolic dysfunction
 - Pericardial effusion

23. INTERVENTION GUIDING ECHOCARDIOGRAPHY

Echo guided pericardiocentesis

1/Confirmation of intra-pericardial needle position

-Agitated saline administrated through pericardiocentesis needle

- 2/Selection of needle entry site
 - -Largest fluid collection closest to the chest wall
- 3/Post procedure assessment

Echo guided myocardial biopsy

1/Advantages

-Wider choice of biopsy sites

-Change biopsy site at follow-up

-Avoid tricuspid valve

2/Methods

-TTE apical or subcostal 4 chambers view (modified for better RV visualisation)

-TOE mid-oesophageal 4 chambers view or transgastric SAX or LAX

3/Post procedure assessment

Echo guided trans-septal catheterisation

1/Advantages

-Visualisation of catheter position relative to the fossa ovalis

-Helps avoid aortic puncture

-Speeds up procedure reducing fluoroscopic time

2/Methods

-Confirm IAS tenting before puncture

-Bubbles study confirmation of position (RA / LA)

-TTE / TOE with 2D / 3D

3/Post procedure assessment

Echo during ASD / PFO closure

1/TOE

-Used in the majority of centres

-Usually under general anaesthesia to minimise risk of aspiration and maximise comfort

-Patient selection for procedure

-Exclude co-existent anomalous venous drainage

-Guide device positioning

-Procedure success assessment (bubbles study)

2/Post procedure echo assessment

Echo guiding left atrial appendage (LAA) closure

1/TOE 2D / 3D

2/Pre-procedure assessment

-Exclude thrombus

-Assess LAA morphology

Ostial dimension

Maximum length of dominant lobe

-Mitral valve and pulmonary veins flow (especially left upper pulmonary vein)

3/Procedure guiding

-Trans-septal puncture

-LAA obliteration (device positioning)

4/Post-procedure assessment

-LAA obliteration success

- -Complications
 - Pericardial effusion
 - Thrombus around the device
 - Mitral valve function
 - Left upper pulmonary vein flow
 - LV function

5/Follow-up

Echo during percutaneous mitral balloon valvotomy

1/TTE

-To guide trans-septal catheterisation, measure gradient before and after, detect MR, detect pericardial effusion

-Can interfere with the procedure / sterile technique

-Limited by echo window -2D / 3D

2/TOE

-Usually under general anaesthesia to minimise risk of aspiration and maximise comfort

-To guide trans-septal catheterisation, detect LAA thrombus, measure gradient before and after, detect MR, detect pericardial effusion, and assess the residual ASD

-Speeds up procedure reducing fluoroscopic time

-2D / 3D

3/Post procedure assessment

The role of echo in transcatheter mitral valve repair

1/Patient selection

-TTE / TOE with 2D / 3D

-Coaptation length / coaptation depth / flail gap / flail width

-Exclusion criteria

-Complex MV disease

-Severe MV deformation

-Rheumatic MV disease

2/MitraClip device deployment guidance – to grasp anterior and posterior leaflet together reducing MR

-TOE 2D / 3D

-Transseptal punctures guidance

-Clip to be positioned just above the orifice (max PISA), perpendicular to the commissures

-Assessment of residual MR / iatrogenic MS / leaflet lesions / failure to grasp

-Detection of complications

3/Post procedure assessment

The role of echo in TAVI

1/Patient selection

-TTE

- Assessment of AS severity
- Assessment of annulus size
- Identification of contraindications

-TOE

- Assessment of aortic root anatomy
- Aortic annulus size
 - Measure in systole
 - In 3D guided 2D both in sagital and coronal planes
- Cusps morphology
- Annulus to coronary ostia distance
- 2/Procedure guiding TOE
 - -Guide balloon positioning
 - -Detect post-valvuloplasty AR
 - -Aid prosthesis positioning

-Check prosthesis function immediately post implantation

-Detect complications

- Prosthesis misplacement
 - Embolisation towards aorta / LV
 - Too high / too low

• AR

- Central
- Para-valvular
- MR
 - Prosthesis impinges on anterior MV leaflet
 - LV dyssynchrony due to RV pacing
 - Damage or distorsion of the subvalvular apparatus by delivery system
- New LV regional wall motion abnormalities
 - Acute coronary ostium occlusion
- Tamponade
- Dissection / rupture of the aortic root
- 3/Post procedure assessment
 - -Valve function
 - -Complications

The role of echo in paravalvular leak closure

- 1/Patient selection
 - -TTE / TOE with 2D / 3D
- 2/TOE 2D / 3D
 - -Quantify regurgitation
 - -Identify defect, demonstrate irregular defect shape, size defect (>25% circumference not for percutaneous closure), detect thrombus -Procedure guidance
 - Guide trans-septal catheterisation
 - Guide device positioning with real time 3D zoom
 - Ensure device does not interfere with mechanical prosthesis function
 - Assess success
 - Detect complications
- 3/Post procedure assessment

Intraoperative echo

- 1/Aortic cannulation site guidance
 - -Detection of atheroma
- 2/Procedure guidance
 - -Valve repair

• MV

- Confirmation of valve morphology and lesion
- Annular dimensions (antero-posterior / commissural), anterior leaflet: annulus ratio
- $\hfill{\circ}$ Post-repair assessment (residual regurgitation / prolapse, induced stenosis or SAM)
- AV
- Confirmation of valve morphology and lesion
- Post-repair assessment (residual regurgitation / prolapse, induced stenosis or aortic dissection)
- TV
- Measurement of annulus (3D)
- Post repair assessment (residual regurgitation, induced stenosis)
- -Infective endocarditis
 - Delineation of damage & assessment of success
- -Prosthetic valves
 - Assessment of procedural success & detection of complications

-Aortic surgery

- Post-surgical assessment (closure of entry site, absence of leak)
- -Myectomy
- -Cardiac masses
- -Pericardial effusion/Haematoma
- -Assist device implantation (V-V device / V-A device)
- 3/Immediate post-procedural assessment
 - -New regional wall motion abnormality
 - -Pericardial effusion
 - -Abnormal inter-cavitary communication / abnormal flow
 - -New valve lesion / new valve regurgitation
- 4/LV and RV function monitoring
- 5/Detection of intracardiac air

24. PRINCIPLES OF QUALITY ASSESSMENT IN ECHOCARDIOGRAPHY

Principles of Quality Measurements

1/Individual accreditation

- -Training
- -Accreditation
- -Re-accreditation
- 2/Laboratory accreditation
 - -Laboratory infrastructure
 - Equipment minimum standard
 - Staff proficiency standard
 - -Patient selection
 - Appropriateness of studies
 - Adequate case mix of pathologies
- 3/Study performance
 - -Diagnostic quality
 - Standardization of performance, storage and reporting
 - Stress-echo policy
 - TOE policy
 - Contrast-echo policy
 - -Patient safety
 - Waiting-list monitoring
 - Waiting list clinical prioritization
 - Monitoring complications
- 4/Study interpretation
 - -Accuracy
 - -Reproducibility
 - -Complete report

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