EAE CORE SYLLABUS
A learning framework for the continuing medical education of the echocardiographers

Prepared by the Education Committee of the European Association of Echocardiography

EAE Education Committee 2009-2010
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Foreword

The Core Syllabus in echocardiography which is provided by the European Association of Echocardiography, represents a major step forward in harmonization of cardiology training in Europe. The European Association of Echocardiography is a world leader in teaching and training in echocardiography, and provides high quality education via congresses, journals, website and other educational products. The Core Syllabus provides a summary of the core knowledge base within echocardiography and is targeted for cardiology fellows and for continuing medical education of trained cardiologists. In addition, the document will be useful for sonographers and physicists involved in clinical echocardiography. Ultimately, the success of the Core Syllabus depends on its adoption at the national level, and we hope it will be used as a framework for trainers and trainees in teaching hospitals across Europe. We also expect it will be used to standardize the content of educational activities within the European Association of Echocardiography and in its external relations with National Societies and National Working Groups on echocardiography. The Core Syllabus is developed with contributions from internationally leading experts in the field and each one should be thanked for their contribution.

Roberto Ferrari  
President ESC  

Otto A. Smiseth  
Chairman ESC Education Committee
Preface

Providing adequate education is one of the main goals of the mission of the European Association of Echocardiography (EAE). Echocardiography is able to provide an impressive amount of information from different modalities (M-mode, two- and three-dimensional, Doppler), approaches (transthoracic-TTE, transesophageal-TEE, intravascular, epicardial), and applications (e.g. stress and contrast echocardiography), but needs an adequate training of operators to be performed in a cost effective and reliable way. The purpose of producing a Core Syllabus is to lay out the range of knowledge that the EAE expect the European echocardiographer to possess. This document represents a driving force for the EAE to deliver educational resources to assist echocardiographers in achieving accreditation and perform comprehensive and accurate echocardiographic studies.

Luigi P. Badano, MD, FESC
EAE President Elect
Chair EAE Education Committee

Prof. Jose Luis Zamorano, MD, FESC, FACC
EAE President
Introduction

Echocardiography is an ultrasound based imaging modality which provides a non-invasive assessment of the structure, function and haemodynamics of the heart through a real time virtual view of cardiac chambers. Echocardiography is a major contributor to the practice of cardiology because it allows to identify cardiac abnormalities and aids management of cardiovascular disease in children and adults. The ability of echocardiography to provide unique noninvasive information with minimal discomfort or risk, without using ionizing radiation, coupled with its portability, immediate availability, and repeatability, explains its use in virtually all fields of cardiology. However, echocardiography remains an operator dependent technique. A thorough knowledge of cardiovascular anatomy and pathophysiology together with appropriate technical skills are required to perform a comprehensive and clinically useful echocardiographic study. 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 echocardiographer should be able 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 clinical data, ultrasonographic image content, and related physiologic knowledge. Knowledge of the principles of ultrasound physics and instrumentation and its continuous application during the examination is prerequisite to obtain optimal data.

The Core Syllabus.

The EAE Core Syllabus is a framework of the core echocardiographic knowledge that an echocardiographer needs to possess. Throughout the document, the word „Echocardiographer“ refers to any operator (i.e. doctors, sonographers, physicists) who intends to use ultrasound for clinical purposes.

The purposes of the Core Syllabus.

The Core Syllabus provides a structure for the educational and accreditation activities of the EAE both internally and in its external relations with other National Societies and/or Working Groups on echocardiography.

Internal use of the Core Syllabus. The EAE Core Syllabus will represent a platform to facilitate a structured approach to CME (Continuous Medical Education) for echocardiographers. The Education Committee will use the document to develop educational courses and products accordingly such as:

- Teaching courses
External use of the Core Syllabus. The EAE Core Syllabus will assist in:

- Standardizing the content and planning of educational events organized by National Societes/Working Groups;
- Planning medical education in Cardiovascular Medicine thereby standardizing 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 EAE Core Syllabus will facilitate the cooperation between EAE Education Committee, ESC Education Committee and EBAC in providing adequate CME credits at various educational programmes run throughout Europe.

Future developments
The Core Syllabus is the first step to develop the EAE Core Curriculum, an objective for this Education Committee over the next 2 years. The Core Curriculum is deemed to be an expansion of the Core Syllabus based on educational objectives. It will specify learning, teaching and assessment methods.
Acknowledgements

We acknowledge the work of the previous EAE Education Committee in preparing the first draft of this document. A special thank to the colleagues who devoted their time and expertise in reviewing the Core Syllabus: Nuno Cardim (Portugal), Frank A. Flachskampf (Germany), Jane Graham (United Kingdom), Aleksandar Neskovic (Serbia), and Jens-Uwe Voigt (Belgium). Their thoughtful review as well as their comments and suggestions have greatly improved the readability and comprehensiveness of the document.
1. GENERAL PRINCIPLES OF ECHOCARDIOGRAPHY

Principles of Ultrasound

Physics of ultrasound

- Sound wave: compression and rarefaction
- Differentiation between audible sound and ultrasound frequency ranges
- Diagnostic frequency range, the trade-off between penetration vs. spatial resolution

Characteristics of an ultrasound wave

- Frequency, relation to wave length
- Amplitude, relation to Power, Intensity, Pressure
- Average speed of sound in tissues

Reflection and transmission of Ultrasound at interfaces

- Acoustic impedance
- Reflection and Refraction
  - Return signal ratio, its dependence on insonation angle and interface acoustic impedance mismatch
- Scattering
  - Return signal ratio, its dependence on scatterer size and frequency

Attenuation

- Sources of attenuation
- Frequency dependence
- Effect on images

Transducers

- Transducer construction and characteristics
  - Piezoelectric element: piezoelectric effect
  - Types: mechanical transducer, 1D-, 1.5D-, Matrix-Array Transducer

Sound beam formation, steering and focusing

- Methods of focusing (curved element, electronic)
- Focal zone characteristics (maximum intensity, depth of focus, focal area)
- Side lobes, influences on image quality
- Methods of steering
- Focussing during sending, multiple focussing on receive side

Transducer selection

- Size and shape
  - Large Transducer has better beam characteristics, sharper focus at deeper depth
  - Small Transducer needs smaller acoustic window, easier to handle
- Frequency
  - trade-off between penetration vs. spatial resolution

Imaging Principles of Ultrasound

- Imaging modes (advantages and limitations)
  - A-mode, B-mode, M-mode
  - 2-dimensional (2D), 3-dimensional (3/4D)
  - Pulsed wave Doppler, continuous wave Doppler

Signal processing

- A/D conversion, No of beams
- Relation between No of beams / Sector with / Depth / Frame Rate

Harmonic imaging

- Principles
- Impact on image quality
- Use in contrast echocardiography

Image Storage
Paper, Video, Digital
DICOM, HL7 principles

Optimizing image quality
Output power
Dynamic range, Compression
Receiver overall gain, Time gain compensation, Lateral gain compensation, Reject

Artifacts and pitfalls of imaging
Reverberations
Aliasing
Mirror images
Near field clutter, Side lobes
Refraction, Shadowing
Stitching artifact (3/4D)
Blooming (contrast)

**Biologic Effects of Ultrasound and Safety**
Dosimetric quantities (Pressure, intensity, power and area)
Factors affecting acoustic exposure, Equipment controls
Biological / Physical effects
Cavitation
Heating

**Quality Assurance of Ultrasound Instruments**
General concepts
Need for quality assurance
Nature of a quality assurance program

**Principles of Doppler Echocardiography**
Physical principles
Doppler effect (as related to sampling red blood cell movement)
Fast Fourier transformation
Doppler equation
Angle of incidence
Colour Doppler Processing

Spectral Doppler
Differences between pulsed and continuous-wave Doppler (pros/cons)
Pulsed wave Doppler
Sample volume(s), Aliasing, Pulse repetition frequency, HPRF
Nyquist frequency limit, Maximum depth, Baseline position

Continuous wave Doppler
High-velocity measurement capability

Characteristics and information of spectral display
Spectral broadening and artifacts

Colour Doppler
Sample volume size, Aliasing, Scale, Maximum depth, Baseline position
Power Doppler principle
Tissue Doppler principle

Characteristics and information of color display
Colour Maps
Variance Display
Postprocessing options
2. THE ECHOCARDIOGRAPHIC EXAMINATION

The Echo Exam

Basic imaging principles
   Technical quality
      Use of equipment controls
      Recognition of technical artifacts
      Recognition of setup errors
   Nomenclature of standard views, Myocardial segmentation
   Image orientation, relation between scan planes, Bulls eye display, Coronary artery territories

Transducer positions and views
   Parasternal
      Long axis of LV
      Short axis of LV
      Right ventricle (RV) inflow and outflow views
   Apical
      4-chamber view
      2-chamber view
      3-chamber view (long axis)
      Other apical views
   Suprasternal notch
   Subxiphoid
   Other acoustic windows

M-Mode echo
   Aortic valve and left atrium (LA)
   Mitral valve (MV)
   LV
   Other M-mode recordings

Principles of echo measurements
   M-mode
   2D echo

Special Techniques
   Use of contrast agents
   Provocative maneuvers

Anatomy and Physiology of the Heart and Great Vessels

Left Ventricle
   Dimensions, area, volumes
   LV mass, wall thickness
   Global and regional systolic function
   Diastolic function (see section on diastolic function)
   Interdependence of LV and RV

Right Ventricle
   Dimensions, area, volumes
   Global systolic function (see section 4)
   Echo findings with RV volume and pressure overload
   Moderator band

Left Atrium
   Dimensions, area, volumes
   LA function

Right atrium (RA)
   Ventricular septum and causes of "paradoxical" septal motion
   Atrial septum
   Left ventricular outflow tract (LVOT)
Pulmonary veins
Inferior (IVC) and superior vena cava (SVC)
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 pulmonary arteries
  Ductus arteriosus Botalli
Coronary sinus
  Normal imaging
  Causes of dilatation
  Differentiation from descending thoracic aorta
Coronary arteries
  Normal imaging
  Doppler flow patterns
  Coronary flow reserve
Mitral valve apparatus
  Leaflets
    Scallops
  Chordae tendinae
  Annulus
    Normal size
    Variability throughout cardiac cycle
    Nonplanar shape
  Papillary muscles
Aortic valve
  Leaflets, commissures, annulus
  Subvalve, supravalve
Tricuspid valve
  Leaflets (anterior, septal, posterior)
  Papillary muscles
Pulmonic valve

**Arrhythmias and Conduction Disturbances**
  Production of wall motion abnormalities (WMA)
  Effect on valve motion
  Effect on Doppler flow velocity waveforms
3. ASSESSMENT OF DIAMETERS, VOLUMES AND MASS

Assessment of Cardiac Diameters

Methods

- M-mode, parasternal long-axis (for LV: only when perpendicular to septum and crossing mitral leaflet tips)
- 2D Imaging or anatomical M-mode (in all other circumstances)

Pitfalls and problems

- Malalignment of M-mode line
- Wrong timing
- Basal hypertrophy
- Right ventricular diameters unreliable

Assessment of LV Volume

Methods

- Biplane method of discs (modified Simpson’s rule)
- Single plane area-length
- Full volume 3D

Technical considerations

- Correct image plane
  - largest dimension of chamber, no foreshortening
  - both AV valves imaged
  - avoid aorta (anterior) and coronary sinus (posterior)
- Selection of precise time in cardiac cycle for measurements

Pitfalls and problems

- Endocardial dropout (especially apical views)
- Foreshortening of LV cavity (will overestimate ejection fraction)
- No correct timing (difficult in left bundle branch block, pacemaker)

Technologies to improve endocardial delineation

- Harmonic images
- Contrast agents

Assessment of Left ventricular volume

End-systolic volume

- Most reproducible volume measurement
- Relatively insensitive to cardiac loading
- Powerful predictor of cardiac events
- Normal values
- Reproducibility ± 15% (95% CI)

End-diastolic volume

- Endocardium more difficult to image at end diastole
- More variable than end-systolic volume
- Normal values
- Reproducibility ± 25% (95% CI)

Assessment of RV Volume

Methods

- No reliable estimate with 2D imaging
- Full volume 3D Echo reliable (image quality permitting)

Left ventricular mass

Method

ASE equation: \[ LVM = 0.80 \times 1.04 \times [(Dd + S + PW)^3] - (Dd)^3 \] + 0.6

LVM = Left ventricular mass
Dd = LV end-diastolic diameter
Pitfalls and problems
   Limited by relatively wide standard deviations
Normal values
Clinical significance
   Prognosis in coronary artery disease, acute myocardial infarction
   Prognosis in Hypertension, Dilated Cardiomyopathy
Therapeutic implications
   Angiotensin converting enzyme inhibitor treatment
   Cardiac resynchronization and/or implantable defibrillator implant
   Timing of surgery in volume / pressure overload
4. NONINVASIVE HEMODYNAMICS DERIVED FROM ECHO-DOPPLER

Basic Principles
- Laminar vs. turbulent flow
- Flow velocity profiles of valves and vessels

Principles of Volume and Flow Measurement
- Principle of stroke volume calculation from Doppler
  \[ \text{Stroke volume (SV)} = \text{Cross sectional Area (CSA)} \times \text{Velocity Time Integral (VTI)} \]
- Application to all four valves
- Potential measurement sites
- Assumptions for area estimations
  - Diameter measurements assuming circular areas
  - Area constant throughout cardiac cycle
  - Area and velocity measured at same site
  - Doppler beam aligned parallel to blood flow
- Limitations, possible measurement errors and reliability
  - less reliable in tricuspid and mitral valve
  - 5 to 10 beats in atrial fibrillation
- Principle of stroke volume estimation from LV volume (2D echo)
  \[ \text{SV} = \text{LV end-diastolic volume} - \text{LV end-systolic volume} \]
- Application to Cardiac output estimation (CO)
  \[ \text{CO} = \text{SV} \times \text{heart rate} \]
- Application to shunt estimates
  - Pulmonary-to-system flow ratio (Qp/Qs)
- Application to Regurgitant volume (RV) and regurgitant fraction (RF) estimation
  \[ \text{RV} = \text{volume of blood that regurgitates through incompetent valve} \]
  \[ \text{Regurgitant Fraction} = \frac{\text{Regurgitant Volume}}{\text{Stroke Volume}} \]

Normal Antegrade Intracardiac Flows
- LV outflow
  - Apical 5-chamber outflow
  - Normal values
- Right ventricular outflow
  - Parasternal short-axis view
  - Sample volume in right ventricular outflow tract or proximal pulmonary artery
  - Normal values
- LV inflow
  - Apical 4-chamber view
    - E wave velocity
    - E wave deceleration time
    - A wave velocity and duration
    - E/A
    - Normal values (for ages 20 to 50 years) at mitral leaflet tips (age-dependent)
- Pulmonary venous flow
  - Apical 4-chamber view
  - Normal values
    - S1
    - S2
    - D
    - S/D
    - A velocity and duration
- Descending aorta flow
- Inferior and superior vena cava, and hepatic veins flow
Coronary arteries
  Left anterior descending
  Right posterior descending
  Circumflex branch
  Coronary flow reserve (use of vasodilator agents)

**Assessment of Intracardiac Pressures**

**Principle of Bernoulli equation**
- Conservation of Energy principle
- Pressure gradient proportional to acceleration
- Poststenotic loss of energy due to turbulence ($p_{stenosis} = p_{behind\ stenosis}$)

**Modified Bernoulli equation** ($P_1 - P_2 = 4 (V_2^2 - V_1^2)$)
**Simplified Bernoulli equation** ($P_1 - P_2 = 4V_2^2$)

**Assumptions**
- $V_1$ is negligible
- Flow through a stenotic orifice (not valid for prostheses)

**Pitfalls**
- Improper beam alignment (large angle $2\theta$)
- Poorly recorded signals (signal-to-noise ratio)
- Failure to detect an eccentric high-velocity jet
- Long, tubular stenoses (viscous friction component becomes significant)
- Changes in viscosity (e.g., anemia, polycythemia)
- $V_1$ may be significant (especially with mild stenosis, regurgitation, high output)

**Pressure recovery**
- reduced poststenotic turbulence allows recovery of potential energy
- echo based gradients higher (pre-stenotic vs. stenosis) than catheter measurements (pre-stenotic vs. post-stenotic)
- 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
- Pulmonary artery diastolic pressure,
  LV diastolic pressure, pulmonary artery pressure/right ventricular systolic/diastolic pressure

**M-mode findings**
- Tricuspid ring IVRT findings
- Pulmonary acceleration time
- Systolic time intervals

**Continuity Equation**

**Basic principle**
- Conservation of mass
- Flow volume before a valve equals flow volume across a valve

**Equation**
\[(\text{Area}_1) \times (\text{VTI}_1) = (\text{Area}_2) \times (\text{VTI}_2)\]

**Application to Aortic valve area estimation**

**Technique**

**Pitfalls**
- Inaccurate LV outflow tract diameter measurement
- Inaccurate LV outflow tract velocity ($V_1$)
- Inaccurate transvalvular velocity ($V_2$ or $V_{max}$)
- Irregular rhythm (e.g., atrial fibrillation); average 8 to 10 beats
Low output states
Distinction between anatomic orifice and effective orifice

Application to Mitral valve area estimation
 Technique
 Pitfalls
 See list for AS assessment plus Aortic regurgitation

**Pressure Half-Time Method**
 Definition of Pressure Half-Time
 Determinants
 orifice area
 Pressure difference
 compliance of involved chambers / vessels
 Application to Mitral valve area assessment
 Equation
 Mitral valve area = 220/T½ (220 = empirical constant)
 Pitfalls
 LV Hypertrophy
 Aortic Regurgitation
 Atrial septal defect

Application to Aortic regurgitation assessment
 Technique
 Pitfalls

**Proximal Isovelocity Surface Area (PISA)**
 Definition and principles
 Flow converges toward a restrictive orifice in a laminar fashion with isovelocity surfaces that approximate hemispheres
 Conservation of mass principle (see Continuity Equation)
 Volume flow across any isovelocity surface = Volume flow through orifice
 Application to Mitral Regurgitation Assessment
 FlowMR = Areashell x Vshell = 2Ar² x Vr
 FlowMR = instantaneous flow rate (cc/s)
 r = radial distance of the isovelocity shell from orifice (cm)
 Vr = flow velocity at radius r (cm/s)
 Effective regurgitant orifice (ERO)
 EROMR = (FlowMR) / VMR
 Average effective area of the regurgitant orifice
 Corresponds to severity of regurgitation
 Regurgitant volume (RV)
 RVMR = EROMR = EROMR x TVIMR

Assumptions
 Advantages
 Can be used in presence of aortic regurgitation
 Quantitative assessment
 Limitations
 Assumption of spherical flow convergence area
 Geometry of isovelocity shells changes with flowrate and pressure gradient
 Flail mitral leaflets may cause a funnel-shaped convergence region (<180°) resulting in overestimation if hemisphere
 Inability to accurately measure radius in some patients
 High wall filter increases Doppler velocities, causing overestimation of flow rate

Application to of mitral valve area (mitral stenosis) assessment
 Mitral valve Area = Flowmitral / Vpeak inflow
Advantages
- Can be used in presence of aortic regurgitation
- Mitral regurgitation does not affect mitral valve area calculation

Limitations
- Same as with mitral regurgitation (above)
- Relatively less well-validated than other methods
- Higher aliasing velocity (>25 cm/s) may tend to underestimate mitral valve area

Other uses
- Aortic Regurgitation
- Atrial and ventricular septal defect shunt flow
- Aortic coarctation area

Contractility Assessment (dP/dt)
Definition
- Approximation of dp/dtmax by measuring the pressure rise at the mitral regurgitation signal between 1 and 3 m/s.

Utility
- Indirect, non-invasive measure of myocardial contractility
- Relatively afterload-independent

Assumptions
- CW Doppler velocity of mitral regurgitation reflects instantaneous peak gradient between LV and left atrium
- Left Atrium is compliant (left atrial pressure stable during pre-ejection period)

Technique, dP/dt values

Pitfalls of dP/dt
- Poor alignment of CW cursor with mitral regurgitation jet (underestimates)
- Acute mitral regurgitation (noncompliant left atrium, left atrial pressure rises with mitral regurgitation)
- Preload-dependent
5. ASSESSMENT OF SYSTOLIC FUNCTION

Determinants of LV Performance

Contractility (inotropic state of myocardium)
End-systolic elastance of ventricle
determined invasively by evaluating ventricular pressure/volume loops at different loading conditions
Preload (Fiber length at onset of contraction)
End-diastolic volume
LV end-diastolic pressure
Afterload (Counter force to contraction)
Ventricular shape and wall thickness
Ventricular systolic pressure
Arterial resistance
Aortic impedance
Mass of blood in aorta
Viscosity of blood

Global LV Systolic Function

Measurements
Ejection fraction
Fractional shortening
Velocity of circumferential fiber shortening
Cardiac output and Stroke volume
Non-ejection phase indexes
Systolic time intervals
dP/dt
Acceleration time
Myocardial strain
Longitudinal AV-valve ring displacement

Determinants
Preload
Afterload
Heart rate, Rhythm

Ejection Fraction
Assessment
Reproducibility ±10%
Visual estimation of left ventricular ejection fraction
Generally valid by experienced echocardiographer
Interobserver variability
Quantitative assessment of left ventricular ejection fraction
Based on LV volume estimates in systole and diastole
Area-length method
Modified Simpson’s rule

Pitfalls (over/underestimation)
Mitral / Aortic regurgitation
Aortic stenosis, severe Hypertrophy
Severe anemia
Bad LV filling, Hemodialysis patients

Fractional shortening (%)
Simple, one-dimensional M-mode echo technique
Should not be used any more
Assessment
1 – end-systolic diameter/end-diastolic diameter
Pitfalls (over/underestimation)
- M-mode line not basal and not perpendicular to the Septum
- Regional myocardial disease
- Velocity of circumferential fiber shortening

**Regional Systolic Function**
- Left ventricular segmentation
  - 16, 17 and 18 segment model
  - Coronary flow distribution and left ventricular segmentation
- Right ventricular segmentation
- Visual Wall motion analysis
  - Endocardial motion
  - Myocardial thickening
  - Scar recognition
  - Definitions
    - Hyper-, Normo-, Hypo-, A-, Dyskinesis
    - Aneurysm
- Wall motion score index
- Quantitative techniques
  - Strain Rate Imaging
  - Border recognition and tracking in 2D / 3D

**Interdependence of LV and right ventricle**
- Alterations in pressure, volume or both in one ventricle affects the function of the other
  - Left and right ventricle share septum
  - Left and right ventricle circumferential myofibers
  - Surrounding pericardium constrains the ventricles within a limited space
- Right ventricular volume overload
  - Ventricular septal flattening and leftward displacement in diastole only
  - Typical clinical conditions
- Right ventricular pressure overload
  - Ventricular septal flattening and leftward displacement in both systole and diastole
  - Typical clinical conditions
- Behaviour at constrictive / restrictive disease
- Diagnostic Maneuvers
  - Müller’s maneuver
  - Valsalva maneuver

**Global Right Ventricular Systolic Function**
- Right ventricular ejection fraction (2D / 3D based)
- Fractional chamber diameter changes
- Fractional Area change
- Tricuspid annular plane systolic excursion (TAPSE)
- Tei index
6. ASSESSMENT OF DIASTOLIC FUNCTION

Basic Principles

Hemodynamic phases of diastole
- Isovolumic relaxation
- Early rapid diastolic filling
- Diastasis
- Late diastolic filling caused by atrial contraction

Physiologic parameters of diastolic function

Relaxation
- active component (breakdown of crossbridges)
- begins mid-systolic, ends with diastasis
- invasively often determined by Time constant of relaxation (Tau)

Compliance
- passive properties of the myocardium and pericardium
- may be pressure / geometry dependent
- invasively mostly determined by the Diastolic pressure-volume-relation

Echo-Doppler Approach to LV Diastolic Function

Parameters to consider
- Chamber dimensions
- Wall thickness
- Mitral E- and A-wave velocity, E/A ratio, A-wave duration
- Isovolumic relaxation time
- Mitral E-wave Deceleration time, deceleration slope
- Pulmonary venous flow (S/D ratio, AR wave amplitude, duration)
- Mitral Ring velocities (E’-wave, E/E’ ratio)
- Mitral inflow colour flow M-mode (E/VP)

Echocardiographic Assessment of LV Diastolic function

Technique of measurements

Hierarchy of measurements

Typical Categories
- Normal function
- Abnormal relaxation
  - Echocardiographic features
  - Clinical appearance, Significance
- Pseudonormal
  - Echocardiographic features
  - Clinical appearance, Significance
  - Distinguishing pseudonormal from normal/LV filling
- Restrictive
  - Echocardiographic features
  - Clinical appearance, Significance
- Irreversible restrictive
  - Echocardiographic features
  - Clinical appearance, Significance
- Elevated Filling pressure
  - Echocardiographic features
  - Clinical appearance, Significance

Pitfalls and Factors that affect Echo Measurements
- Sample volume location, Intercept angle
- Respiration, Valsalva Maneuver
- Heart rate, Rhythm
- Preload, Afterload, Exercise
LV systolic function and end-systolic volume
Atrial function, volume and compliance
Mitral stenosis, relevant regurgitant lesions
Clinical applications (conditions associated with diastolic dysfunction)
7. ISCHEMIC HEART DISEASE

Coronary Anatomy and Function
Coronary arteries and corresponding myocardial territories
Anomalous origin or course
Coronary aneurysms (echo findings)
Coronary fistulae (echo findings)
Normal coronary sinus and malformations
Coronary atherosclerosis

Myocardial Ischemia
Pathophysiology
Ischemic cascade
Relation of wall motion and wall thickening to coronary artery perfusion
Detection of ischemia
Reduced Endocardial motion
Reduced thickening/shortening
Post-systolic shortening
Diastolic function changes
Quantitative assessment (Strain Rate Imaging)
Pitfalls and Limitations
Translational motion, Through plane motion
Conduction or pacing abnormalities
Role of Stress testing for ischemia (see stress echo)

Myocardial Infarction
Detection of myocardial infarction
Early / Late appearance, Scar
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
Complications and associated findings
Acute ischemic mitral regurgitation
Free wall rupture
Ventricular septal rupture
Aneurysm, pseudoaneurysm
Papillary muscle rupture
Right ventricular infarction
Left ventricular thrombus
Infarct expansion and extension
Follow-up
Remodeling (infarct expansion, global dilatation)
Recovery of function
LV thrombus
Ischemic mitral regurgitation
8. HEART VALVE DISEASES

Aortic stenosis
Etiology
  Congenital
    Bicuspid aortic valve
    Unicuspid aortic valve
    Association to Coartation
  Rheumatic
  Degenerative (calcific)
Quantitation
  Pressure gradients
  Valve Aerea
    Continuity equation
    Planimetry
  Valve resistance
Pitfalls and Problems
  Low cardiac output
    Gradient underestimates severity
  Low-gradient aortic stenosis
    Explanations
      Role of stress echo in order to differentiate
    LV function may be overestimated due to hypertrophy
  Pressure recovery
  LV remodelling
    Aortic root dilatation, assessment of aortic root
Role of hemodynamic stress testing
  Dobutamine echo for low gradient/low LV function aortic stenosis
  Exercise echo in asymptomatic patients with severe aortic stenosis
  Exercise echo in symptomatic patients with moderate aortic stenosis

Pulmonic Stenosis
Etiology
2D Echo findings
  Right ventricular remodelling (hypertrophy, trabeculation, moderator band, D-shaped septum)
Quantitation
  Pressure gradients
  Valve Aerea
    Continuity equation

Sub-/ Supravalvular stenosis
Etiology
  Membrane
  Fibromuscular ridge
2D Echo findings
  Turbulence in Colour Doppler
Assessment
  Pressure gradients
Pitfalls
  Distinction from valvular stenosis

Mitral Stenosis
Etiology
  Rheumatic
Mitral annular calcification and calcific mitral stenosis
Congenital
Miscellaneous
  Myxoma, other tumors
  Cor triatriatum
2D Echo findings at the mitral valve
  Leaflet thickening, especially tips
  Commissural fusion
  “Doming” pattern of leaflets, Funnel shaped apparatus
  Calcification (leaflets, commissures, chordae, annulus, papillary muscles)
  Chordal thickening, fusion (may obliterate secondary orifices)
Quantitation
  Pressure gradients
    Peak (initial gradient)
    Mean gradient
  Mitral valve area
    Planimetry (2D and 3D)
    Pressure half-time method
    Continuity equation
    PISA method
  Technical considerations and pitfalls of each method
Consecutive changes
  Remodelling of cardiac chambers
  Pulmonary hypertension
  Thrombi in left atrium and left atrial appendage
Role of hemodynamic stress testing
  Exercise echo in patients with discrepancy between symptoms and resting hemodynamics
  Exercise echo in patients with sedentary lifestyle (evaluate exercise tolerance, heart rate, blood pressure)
Significant findings
  Rise in mean transmitral gradient (to >15 mm Hg)
  Rise in pulmonary artery systolic pressure (to >60 mm Hg)
Role of echo in percutaneous mitral balloon valvotomy/plasty
  Patient selection (suitability for percutaneous balloon mitral valvotomy)
    Mitral valve Wilkins score (based on morphology of MV apparatus)
    Assessment of anatomy and function
  Echo guidance during valvotomy
    transseptal puncture, balloon positioning
    Immediate assessment of results / complications
Indications for TEE

Tricuspid Stenosis
Etiology
  Rheumatic
  Congenital
  Carcinoid
  Fabry’s disease
  Previous methysergide therapy
Quantitation
  Pressure gradients
    Peak, Mean gradient
  Tricuspid valve area
    Continuity equation
    Planimetry (3D)
Technical considerations and pitfalls of each method
Other cardiac abnormalities

**Basic Principles of Valve Regurgitation**

**Fluid dynamics**
- Regurgitant orifice (size, shape)
- Proximal flow acceleration
- Vena contracta
- Flow disturbance into low-pressure receiving chamber
- Increased antegrade volume flow across valve

Factors that affect 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

**Detection of valve regurgitation**

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

**Direct methods**
- Pulsed Doppler
- CW Doppler
- Color Doppler imaging

**Valvular regurgitation in normal individuals**

**Quantitation of regurgitation severity**

**Semiquantitation**
- Flow mapping (Colour Doppler)
- CW Doppler signal intensity
- Distal flow reversals

**Quantitative**
- Volume flow at 2 sites
- Proximal isovelocity surface area

**Aortic regurgitation**

**Etiology**
- Leaflet abnormalities
- Congenital abnormalities (unicuspid, bicuspid)
- Degenerative valve disease (fibrosis/sclerosis)
  - Rheumatic valve disease
  - Endocarditis
  - Miscellaneous other entities
- Aortic root abnormalities
- Hypertension
Bicuspid valve
Annuloaortic ectasia
Marfan syndrome
Aortic dissection
Miscellaneous other entities

Acute Events
aortic dissection, trauma
infective endocarditis
post–balloon valvotomy or surgical commissurotomy for congenital AS

Indirect signs of aortic regurgitation
Left ventricular dilatation and sphericity
Left ventricular hyperkinesis (until late)
Increased E-point septal separation
High-frequency fluttering of anterior mitral leaflet does not correlate with severity
“Reverse doming” of anterior mitral leaflet (posteriorly displaced anterior mitral leaflet)
Jet lesion on septum or anterior mitral leaflet
Premature aortic valve opening
Increased left ventricular outflow tract velocity
Diastolic mitral regurgitation

Severity of aortic regurgitation
Flow mapping (Colour Doppler)
Jet length, height, area, ratio of jet height/left ventricular outflow tract width
Limitations
  Semiquantitative
  Dependent on physiologic and technical factors
CW Doppler signal intensity
Intense CW signal indicates large regurgitant volume
Limitations
  Semiquantitative
  Dependent on physiologic and technical factors
Aortic regurgitation Pressure Half Time and Shape of CW Doppler curve
Rapid decline indicates severe aortic regurgitation
Limitations
  Semiquantitative
  Poor quality tracing
  Affected by other factors
    Compliance of LV, aorta
    Stroke volume, Afterload
Holodiastolic flow reversal in descending thoracic and abdominal aorta
Conditions and Limitations
Volume flow at 2 intracardiac sites
  limited reliability
Proximal flow convergence method
difficult, limited clinical validation
Diastolic mitral regurgitation
Lacks sensitivity in chronic aortic regurgitation
Premature closure of mitral valve
Lacks sensitivity in chronic aortic valve, not specific

Role of echo-Doppler in timing of surgery
Echo predictors of surgical outcome
Left ventricular end-systolic volume/diameter
Left ventricular end-diastolic volume/diameter
Left ventricular systolic function (ejection fraction)
Rate of ↑ end-systolic size and ↓ ejection fraction over time
Mitral regurgitation

Ethiology
- Rheumatic
- Mitral valve prolapse
- Ruptured chordae
- Infective endocarditis
- Ischemia, infarction
- Dilated cardiomyopathy
- Hipertrophic cardiomyopathy
- Calcified mitral annulus
- Löffler’s
- Connective tissue disorders
- Trauma
- Congenital
- Appetite suppressants

Mechanisms
- Functional classification of Carpentier
  - Normal leaflet motion
  - Excessive leaflet motion
  - Restricted leaflet motion
- Specific mechanisms
  - Annular dilatation
  - Elongated and/or ruptured chordae
  - Abnormal shape/geometry of LV and abnormal papillary muscle orientation
  - Increased rigidity of leaflets
- Jet direction as a clue to mechanism

Indirect signs of mitral regurgitation
- LV dilatation
- Left atrial dilation
- Increased motion of aortic root on M-mode

Severity of mitral regurgitation
- Qualitative
  - Flow mapping
  - CW Doppler signal intensity
  - Increased antegrade velocity caused by increased transmitral volume flow
  - Systolic flow reversal in pulmonary veins
- Quantitative
  - Volume flow at 2 intracardiac sites
  - PISA
    - Instantaneous flow
    - Effective regurgitant orifice area

Chronic vs. acute mitral regurgitation

Sequential evaluation in chronic asymptomatic mitral regurgitation
- Every 6 to 12 months
- Assess changes in LV systolic function
- Assess LV end-systolic size (and/or volume)

Role of echo-Doppler in timing of surgery
- Echo predictors of surgical outcome
- Feasibility of mitral valve repair
- Pulmonic hypertension

Role of hemodynamic stress testing (exercise)
- Patient with mild or moderate mitral regurgitation but exercise-induced symptoms
- Patient with severe mitral regurgitation and minimal or no symptoms
Mitral valve repair
Preoperative evaluation: feasibility of repair
High likelihood of repair
  Ruptured cord to posterior leaflet (especially middle scallop)
  Small perforation
Lower likelihood of repair
  Valve calcification
  Annulus calcification
  Rheumatic involvement
  Ischemic mitral regurgitation
  Anterior leaflet involvement
Intraoperative evaluation of mitral valve repair
  OP success, residual mitral regurgitation / prolapse
  Systolic anterior motion (systolic anterior motion) of anterior mitral leaflet
  New mitral stenosis
Methods of repair

Mitral valve Prolapse
Definition
M-mode
≥ 2 mm posterior displacement of one or both leaflets in mid-late systole, or
Holosystolic posterior “hammocking” ≥ 3 mm
2D echo
  Systolic displacement of one or both leaflets in PLAX view
  coaptation is on atrial side of annular plane
  Distinction to “Flail” mitral valve leaflet(s)
Classification of MV prolapse
Primary
Secondary
  Reduced LV dimensions (e.g. atrial septal defect, hypertrophic cardiomyopathy, pulmonary hypertension, dehydration, straight-back syndrome/pectus excavatum)
  Rheumatic heart disease
  Coronary artery disease (papillary muscle elongation)
Echo findings
  Leaflet thickening (especially if >5 mm)
  Leaflet redundancy
  Enlarged mitral annulus
  Elongated chordae tendinae
  Mitral regurgitation often eccentric (opposite of involved leaflet)
  Mitral regurgitation may be late systolic
Role of echo
  Diagnosis of mitral valve prolapse
  Quantitation of mitral regurgitation
  Risk stratification
  Detection of associated lesions (e.g., ASD)

Tricuspid regurgitation
Ethiology, Imaging of the valve apparatus
  Annular dilatation
  Rheumatic valvulitis
  Carcinoid
  Ebstein’s anomaly; other congenital
  Endocarditis
  Trauma
Radiation therapy
Marfan syndrome
Tricuspid valve prolapse
Papillary muscle dysfunction

Indirect signs of tricuspid regurgitation
Right ventricular and right atrial dilation
Paradoxical septal motion
Right ventricular volume overload

Severity of tricuspid regurgitation
Flow mapping (pulsed or color)
Systolic flow reversal in inferior and superior vena cava
CW Doppler signal intensity

Tricuspid regurgitation jet method to estimate pulmonary artery pressure may be unreliable if no “restrictive orifice” (some cases of severe tricuspid regurgitation)

**Pulmonic Regurgitation**

Ethiology, Imaging of the valve
- Congenital disease
- Endocarditis
- Carcinoid

Severity of pulmonic regurgitation
- Colour Flow mapping (may be missed with too high scale settings)
- CW Doppler intensity
- Shape of CW Doppler time-velocity curve
- Holodiastolic flow reversal in MPA

Clinical utility
- Decision-making in congenital heart disease
- Estimation of PA diastolic pressure

**Prosthetic Valve**

Types of prosthetic valves
Normal Doppler findings
- Antegrade flow patterns
- Physiologic regurgitation
- Prosthetic valve “clicks”

Pathology
- Valve dysfunction
  - Prosthetic valve obstruction
  - Prosthetic valve regurgitation
    - valvular
    - Periprosthetic

Other complications
- Thrombosis, thromboembolism
- Infection
- Pannus (fibrous tissue in growth)
- Dehiscence

Echo-Doppler clues to prosthetic valve dysfunction
- Increased antegrade velocity across valve
- Decreased valve area (continuity equation or pressure half time)
- Increased intensity of CW Doppler regurgitant jet
- Progressive chamber dilation
- Recurrent or unexplained pulmonary hypertension
- Flow convergence on LV side of MV

“Routine” follow-up of prosthetic valve function
Technical aspects and limitations
Acousting shadowing ("flow-masking")
Reverberations
Overestimation of transvalvular pressure gradients
Pressure recovery phenomenon (Especially small size bileaflet mechanical values)

Endocarditis
Diagnosis
Echo hallmark: vegetation
Echo features of vegetation
- Localized echo density (mass)
- Typically irregular shape
- Pedunculated or sessile
- Rarely impair valve motion
- Often flutter or vibrate
- Secondary jet lesions
Location of vegetations
- Usually “upstream” of the valves
- Pacemaker wires
Unusual sites of vegetations
- Chordae tendinae
- Mural endocardium, Mural thrombus
- Eustachian valve
- Calcified mitral annulus
Diagnostic accuracy of echo
- 2D echo (TTE)
- Transesophageal
- 3D (TTE, TEE)
Mimics of vegetations
- Myxomatous degeneration
- Ruptured or redundant chordae
- Focal, nonspecific thickening or calcium deposits
- Retained mitral leaflets/apparatus after MV replacement
- Lambi’s excrecences and valve “strands”
- Sutures, strands on prosthetic sewing rings
- Tumors, thrombi

Hemodynamic sequelae

Prognosis
- Congestive heart failure, death, need for surgery, embolism
- Size of vegetation and risk of embolism
- Morphological score and risk of embolism

Imaging Complications
- Paravalvular abscess
- Intracardiac fistulae
- Valve aneurysm, flail, leaflet rupture
- Aneurysms of mitral aortic intervalvular-fibrosa region
- Dehiscence of prosthetic valve
- Obstruction from bulky vegetation (rare)
- Purulent pericarditis

Special considerations
- Natural history of vegetations
- Active vs. healed vegetations
- Nonbacterial thrombotic endocarditis
- Infections of pacemaker and catheters
Surgery for endocarditis: role of echo
Indications
Timing of surgery
Intraoperative echo
Indications for transesophageal echocardiography

Valvular Heart Disease Associated with Systemic Conditions
Connective tissue diseases
Systemic lupus erythematosus
  Unclear prevalence (varies 10% to 100%)
  Anatomic and functional abnormality usually mild, often clinically silent
  Valve disease does not correlate with clinical features of systemic lupus erythematosus
  Echo findings
    Leaflet thickening (fibrosis)
    Valve masses (Libman-Sacks disease vegetations)
    Valve regurgitation
    Valve stenosis (rare)
Ankylosing spondylitis
  Epidemiology and clinical data
  Echo findings
    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
9. CARDIOMYOPATHIES

Dilated cardiomyopathy

Echo features
Associated findings
Mitral regurgitation
Thrombi
Other chamber enlargement
Pulmonary Hypertension

Prognostic role of echo
Ejection fraction
E wave deceleration time
Right ventricular function

Role of Echo for cardiac resynchronization therapy candidate selection
LV Ejection Fraction
Assessment of Dyssynchrony
Visual (apical rocking)
Quantitative
Timing of Myocardial and/or Mitral Ring Velocities
Apical transverse motion
Septal flash

Limitations
Echo Assessment has no proven additional prognostic value

Therapy guidance
Optimization of pace-maker settings

Hypertrophic cardiomyopathy

Morphologic features
Varied patterns of myocardial hypertrophy (pleomorphic)
Genotype-phenotype correlations
Nondilated left ventricular cavity
Narrowed left ventricular outflow tract diameter
Finely granular speckled appearance of myocardium
Mitral apparatus
Anterior displacement of mitral apparatus
Increased area of anterior mitral leaflet
Atrial dilatation (especially left atrium)

Pathophysiologic features
Systolic anterior motion (SAM) of mitral leaflet (obstructive/non obstructive hypertrophic cardiomyopathy)
Mechanism(s)
Venturi effect (high outflow velocities in narrowed tract)

Other situations
Mitral valve repair
Aortic valve replacement (Aortic Stenosis)
Hypovolemia
Endogenous and exogenous catecholamines

LV out-flow tract obstruction (dynamic)
Narrowed LV outflow tract SAM-septal contact
Increased with certain maneuvers (Standing, Valsalva, Amyl nitrate)
Typical Doppler signal shape (late-peaking)
Upper septal endocardial thickening (“contact lesion”)
Mid-systolic closure of aortic valve

Mid-cavity muscular obstruction (dynamic)
Aliasing begins more apically
Typical Doppler signal shape (late-peaking)
May be induced by dobutamine
Mechanism: Concentric LVH with hyperdynamic contractility
Mitral regurgitation
almost always seen with obstructive systolic anterior motion
Eccentric (posterolateral), late-systolic
Diastolic dysfunction
Diagnostic criteria in first relatives
Limitations and pitfalls of echo
Hypertrophic cardiomyopathy can be mimicked (Chronic hypertension, Cardiac amyloid, Pheochromacytoma, Friedreich’s ataxia, Inferior myocardial infarction with previous left ventricular hypertrophy), Athlete’s heart
Dynamic left ventricular outflow tract obstruction not specific
Apical HCM sometimes missed
False-positive diagnosis because of RV papillary muscle, moderator band, and/or prominent RV trabeculations overlying ventricular septum measurements.
Role of echo in HCM therapy guidance
Medical treatment
DDD pacing (Placement of pacemaker lead, Optimization of AV interval)
Alcohol septal ablation (Patient selection, Guidance of procedure, Follow-up)
Surgical myotomy/myectomy (Determine site and extent of resection, Assess immediate results, Detect/exclude complications)

Restrictive cardiomyopathies
Causes
Primary
Idiopathic
Löffler’s eosinophilic endomyocardial disease
Endomyocardial fibrosis
Secondary
Amyloid heart disease
Hemochromatosis
Heart muscle disease occasionally presenting with restrictive features
Post-irradiation heart disease
Carcinoid heart disease
Doxorubicin/daunorubicin toxicity
Progressive systemic sclerosis
Typical 2D echo findings
Small to normal left ventricular cavity size
Often thickleft ventricular walls
Normal, near-normal LV function
Dilated atria
Doppler: restrictive pattern
Differentiating restrictive cardiomyopathy vs. constrictive pericarditis

Infiltrative cardiomyopathy (overlaps with restrictive cardiomyopathy)
Classification
Interstitial
Amyloid
Hemochromatosis
Sarcoid
Malignancy
Storage
Glycogen storage
Lipid storage
Echo features (used with specific disease)

Arrhythmogenic right ventricular dysplasia
Definition
Primary cardiomyopathy of unknown cause
Characterized by progressive loss of right ventricular myocardium with replacement by peculiar fatty or fibro-fatty tissue
Associated with ventricular arrhythmias and sudden death in young
2D echo findings
Dilated right ventricle
Aneurysms, outpouchings of right ventricle (distributed in RV inflow, apex, infundibulum)
Focal right ventricular wall thinning
Abnormal global, regional right ventricular systolic wall motion
Abnormal tissue composition; right ventricular muscle replaced by fat (better-detected by Magnetic Resonance Imaging)
Lesser involvement of left ventricle (until late)

Other Myocardial Diseases
Myocardial disease associated with neuromuscular disorders
Myocardial disease caused by toxic agents and infectious diseases
Cardiac abnormalities resulting from trauma

Effect of systemic illnesses on the heart
Anemia
Connective tissue disorders
Thyroid disorders
Hemochromatosis
Others
Takotsubo cardiomyopathy
Acute, stress-induced LV dysfunction
Women, mostly elderly
Echo findings
Reversible, balloon-like apical wall motion abnormalities with hyperkinetic base (“apical ballooning”)
Segmental wall motion abnormalities in multiple coronary “territories”
Typical complete recovery in few weeks

Cardiac transplantation
Normal morphologic characteristics and function of allograft
Diminished septal motion and thickening
Exaggerated systolic posterior wall motion/thickening (M-mode)
Increased LV mass
Biatrial enlargement (donor plus recipient)
Biatrial anastomoses enhanced echogenicity
Suturelines (especially noted in apical 4-chamber view)
May be prominent (mass-like appearance)
Right ventricular dimensions
Normal, if pulmonary artery pressures are normal
Dilatation, if pulmonary hypertension preoperatively or perioperatively
Impaired relaxation
Pericardial effusion (small effusion common; “small heart goes in large space”)
Doppler findings in normal allograft
Isovolumic relaxation time and mitral pressure half-time may be short immediately after transplant and increase within 6-weeks
Impaired relaxation
Insignificant valve regurgitation

Potential complications
Acute rejection

2D Echo findings
Increase in left ventricular mass
Decrease in left ventricular systolic function
Increase in myocardial echogeneity
New or increased pericardial effusion

Doppler findings
Decrease in mitral pressure half time
Decrease in LV isovolumic relaxation time
Increase in early diastolic filling velocity (mitral E wave)
New onset of MR

Transplant coronary artery vasculopathy
Stress echo
Coronary flow reserve

Pericardial effusion
Right ventricular systolic dysfunction
Injury to tricuspid valve (second-degree repeated right ventricular biopsies)

Limitations
Alterations in heart rate and loading conditions
Variable timing of recipient and donor atrial contraction
Intersubject and interstudy variation
“Restrictive physiology” early after transplantation

Echo guidance of right ventricular biopsies
10. SYSTEMIC AND PULMONARY HYPERTENSIVE HEART DISEASE

Systemic Hypertension
Physiology, hemodynamics
Increased afterload leads to ventricular hypertrophy and increased mass
Increased hypertrophy/mass leads to diastolic dysfunction
Echocardiographic findings
Increased left ventricular mass and mass index
Hypertrophy
Enlarged left atrium (caused by increased LV diastolic pressure)
Dilated aortic root
Mitral annulus calcification
Right ventricular hypertrophy
Diastolic abnormalities
Role of Echo
Diagnosis, prognosis, efficacy of medical therapy (regression of hypertrophy)
Complications of hypertension
Aortic regurgitation
Aortic dissection
Rule out secondary hypertension (coarctation)
Hypertensive hypertrophic cardiomyopathy of the elderly

Pulmonary Heart Disease (Cor Pulmonale)
Physiology, hemodynamics
Role of echocardiography
Detection of pulmonary hypertension
Detection of occult pulmonary hypertension (exercise echo)
Quantitation of pulmonary hypertension
Determine cause and effects of pulmonary hypertension
Determine prognosis
Assessment of pulmonary hypertension
Distinction of chronic vs. acute cor pulmonale
Acute pulmonary hypertension (acute pulmonary embolism)
Acute right ventricular pressure overload (e.g. pulmonary embolism)
Echo findings
Right ventricular dilatation and dysfunction, right atrial dilatation
Pulmonary artery dilatation
Right ventricular pressure overload of interventricular septum
Thrombus in right heart and/or in pulmonary artery (residual or “in-transit”)
60/60 sign, McConnel sign (specific, insensitive)
May cause exaggerated respiratory variation in mitral and tricuspid inflow (i.e., may mimic cardiac tamponade)
Identification of high risk patients
Right ventricular dysfunction (even in patients without hypotension!)
Free-floating right heart thrombus
Patent foramen ovale
Monitoring the effect of treatment
Chronic pulmonary hypertension (chronic cor pulmonale)
Sustained elevation of pulmonary artery pressure (mean >25 mm Hg at rest or >30 mm Hg with exercise)
2D Echo findings
Right ventricular hypertrophy, dilation
Abnormal right ventricular systolic function
Right ventricular pressure overload pattern of interventricular septum
Pulmonary artery can be dilated
Right atrial dilation, Inter-atrial septum bows Right → Left
Right → Left shunt with contrast (patent foramen ovale)
Pericardial effusion may be present

Doppler findings
Tricuspid regurgitation
Pulmonic regurgitation
Reversal of mitral E/A ratio
11. PERICARDIAL DISEASE

Normal Pericardial Anatomy

Pericardial Effusion
- Detection of pericardial effusion
- Differentiation between pericardial and pleural effusion
- Differentiation between pericardial effusion and epicardial fat
- Quantitation of pericardial fluid
- Echo-Doppler diagnosis of cardiac tamponade
  - Right atrial systolic collapse
  - Right ventricular diastolic collapse
  - Reciprocal changes in ventricular volumes
  - Respiration variation in right ventricular and LV diastolic filling
  - Plethora of inferior vena cava
- Echo-guided pericardiocentesis

Constrictive Pericarditis
- Pathophysiology
- Echo-Doppler diagnosis of pericardial constriction
  - M-mode and 2D echo
    - Pericardial thickening
    - Normal LV size and contractility
    - Atria normal or enlarged
    - “Flattened” motion of posterior wall in diastole
    - Abrupt posterior motion of ventricular septum in early diastole (septal “bounce”)
    - Dilated inferior vena cava and hepatic veins
    - Premature diastolic opening of pulmonic valve
  - Doppler
    - Prominent "Y" descent on hepatic vein or superior vena cava flow pattern
    - LV inflow shows prominent E wave with rapid early diastolic deceleration slope
    - Increase in LV Isovolumic relaxation time by >20% on first beat after inspiration
    - Respiratory variations withincrease in right ventricular filling >40% and decrease in LV filling by 25%
    - Pulmonary venous flow: systolic as often or more than diastolic flow with respiration variation
    - Hepatic vein: decrease or loss of diastolic filling with marked expiratory reversal
- Differential diagnosis versus restrictive cardiomyopathy
  - Pitfalls in differentiating restrictive cardiomyopathy vs. cor pulmonale
    - Chronic obstructive pulmonary disease (false-positive)
    - Increased respiratory effort (false-positive)
    - High filling pressure, Localized constriction (false-negative)
    - Sample volume placement
    - Atrial fibrillation, other irregular rhythms

Pericardial Cysts
- General
  - asymptomatic, benign developmental abnormality
  - Portion of parietal pericardium is disconnected from pericardial sac (no communication)
  - Rare incidental finding
  - Need to be differentiated from other masses
- Echo findings
  - Round or elliptical echo-free space adjacent to a cardiac chamber
**Congenital Absence of Pericardium**

Uncommon congenital abnormality, more men

Total absence of pericardium

Benign

Echo features: variable

- Unusual echo windows (grossly apparent); shift of heart to left chest
- Cardiac hypermobility (exaggerated cardiac motion)
  - *Apparent* right ventricular enlargement (parasternal windows) (Malposition of the right ventricle causes scan plane to transect larger portion of right ventricle)
  - Abnormal ventricular septal motion (mimics right ventricular volume overload)

Recommend multiple imaging modalities to confirm diagnosis

Partial absence of pericardium (less common)

- Potential for herniation, strangulation of portion of heart
- Echo features: variable
  - depending on which portion of heart involved
12. BASICS IN CONGENITAL HEART DISEASE IN THE ADULT

Basic Embryology
- Primitive cardiac formation
- Comparison of fetal and postnatal circulation

Segmental Approach
- Cardiac position
  - Position (in chest)
  - Orientation (position of cardiac apex)
    - Levocardia
    - Dextrocardia
    - Mesocardia

- Visceral situs
  - Solitus
  - Inversus
  - Ambiguous

- Atrial situs (Right atrium right- or left-sided; Left atrium right- or left-sided)
  - Right atrial characteristics
    - Inferior and superior vena cava drain into right atrium
    - Eustachian valve often seen
    - Wide spade-like right atrial appendage
  - Left atrium characteristics
    - Pulmonary veins drain into left atrium
    - Overlap of septum primum onto the superior atrial septum occurs on left atrial side
    - Long finger-like left atrial appendage with narrow neck

- Determine ventricular morphology
  - Right ventricular morphology
    - Triangular shape
    - Coarse trabeculations
    - 3 papillary muscles
    - Moderator band
    - Tricuspid valve attachment
  - Left ventricular morphology
    - Elliptical shape
    - Smooth, fine trabeculations
    - 2 papillary muscles
    - Mitral valve attachment

- Atrioventricular valve identification
  - Mitral valve
    - 2 leaflets
    - 2 papillary muscles
      - Inserts more superiorly on septum
  - Tricuspid valve
    - 3 leaflets
    - 3 papillary muscles
      - Inserts more apically on septum

- Delineate atrio-ventricular connections
  - Atrio-ventricular concordance / discordance
  - Atresia
  - Double inlet
  - Straddling
  - Straddling/override
  - Common inlet
Ventricular topology
  L-looped
  D-looped

Identify great arteries and ventriculoarterial connection
  Vessel area concordance / discordance
  Double outlet
  Great artery position (relative)

Contrast echocardiography for further differentiation
  Agitated saline solution
  Right-to-left shunt
  Systemic venous return anomalies
  Pulmonary arterio-venous fistula

Outflow Obstruction
  Left ventricular outflow tract
    Valvular aortic stenosis
    Subvalve aortic stenosis
      Associated conditions
        Supramitral ring
        Coarctation
      Regrowth after resection
    Supravalve Aortic stenosis
      Associated with Williams syndrome
    Coarctation of the aorta
      Suprasternal view for anatomy and presence of turbulent flow
      Doppler: measure $V_{\text{max}}$ and diastolic flow pattern
      Determine LV dimensions, LV function and hypertrophy
      Associated with bicuspid valve and ascending aortic aneurysm
    Post-repair
      Recoarctation (Bernoulli gradient overestimates severity because of low $V_2$)
      Aneurysm of repair site (Magnetic resonance may be better)

Right ventricular outflow tract obstruction
  Valvular
  Subvalve (infundibular)
  Supravalve
  Associations
    Tetralogy of Fallot
    Williams syndrome
    Noonan syndrome
    Ventricular septal defect
    Arteriohepatic dysplasia
    Peripheral (branch) stenosis
    subaortic stenosis
  Echo-Doppler evaluation
    Document level(s) of obstruction
    Quantify severity of obstruction
    Identify associated abnormalities
  Postintervention (balloon valvuloplasty or surgery)
    Residual or recurrent obstruction
    Pulmonic regurgitation
    Deterioration of RV function

Abnormal Intracardiac Communications
  Atrial septal defect
Types (Secundum, Primum, Sinus venosus, Coronary sinus)
Associated abnormalities
Pathophysiology, hemodynamics, Eisenmenger reaction
2D echo findings
  Signs of right ventricular volume load, sometimes pressure overload
Doppler findings
  Flow begins in early systole, continues almost through cardiac cycle, with a broad peak in late systole and early diastole
  Pulmonary artery pressure elevated (from tricuspid regurgitation signal)
  Qp/Qs (relevance >1.5)
  Colour Mapping
  Contrast
Indications for TEE
Additional Information with 3D
Findings after atrial septal defect repair

Ventricular septal defect
Types
Physiology, hemodynamics, Eisenmenger reaction
Associated lesions
2D echo
  Type and size of defect
  Ventricular size and function (increased with large shunt)
  Left atrial size (increased with large shunt)
Doppler
  Interventricular gradient
  Qp/Qs
  Pulmonary artery pressure
  Colour (Shunt flow, Other abnormalities)
Findings after ventricular septal defect repair

Patent ductus arteriosus
Physiology, hemodynamics
2D echo findings
  LV volume overload
  Dilated left atrium
Doppler
  Specific views (high short axis, suprasternal, high parasternal)
  Pulmonary artery pressure (functional significance)
  Spectral recording of ductal flow signal to assess pulmonary artery pressure (Normally continuous flow with systolic peak)
  Qp/Qs
Associated lesions (patent ductus arteriosus is usually an isolated lesion)
Findings after patent ductus arteriosus repair

Atrioventricular septal defects
Types: Partial / Complete
Associated lesions
Findings after surgery
Partial anomalous pulmonary venous drainage
Physiology, hemodynamics, Common connections
Associated lesions

Persistent left superior vena cava
Echo findings
  Markedly dilated coronary sinus, best seen in parasternal long axis
  Suprasternal view shows left superior vena cava
Left arm contrast injection shows up in coronary sinus first, then right atrium

**Ebstein’s Anomaly**
- 2D echo findings
  - Apical displacement of septal and posterolateral tricuspid valve leaflets into the right ventricle
  - Resultant “atrialization” of right ventricular inflow to varying degrees
  - Enlargement of right atrium
  - Varying impairment of right ventricular function
  - Varying impairment of LV function
- Doppler findings
  - Tricuspid regurgitation
  - Shunt at atrial
  - Right ventricular inflow or outflow tract obstruction

**Associated lesions**
- Role of Echo in patient selection for surgery
  - Determine severity
  - Potential for surgical repair (Mobility and Insertion of anterior leaflet)
  - Presence of atrial communication

**Post-repair assessment**

**Tetralogy of Fallot after Repair**
- Pathophysiology, repair procedure
- Post-surgical evaluation
  - Residual right ventricular outflow tract obstruction
  - right ventricular outflow tract aneurysm
  - Pulmonic regurgitation
  - Assessment of right ventricular size, function
  - Dilated tricuspid valve annulus, tricuspid regurgitation
  - Diameter of main pulmonary artery, left and right pulmonary arteries
  - Residual ventricular septal defect

**Congenital Abnormalities of the Aorta**
- Coarctation of aorta
13. MASSES, TUMORS AND SOURCES OF EMBOLISM

**Infectious Masses**
Vegetation from infective endocarditis

**Noninfectious vegetations** (nonbacterial thrombotic endocarditis)

**Thrombi**

LV thrombus
- Predisposing conditions *(underlying wall motion analysis)*
  - Apical akinesis (especially acute anterior myocardial infarction)
  - LV aneurysm
  - Diffuse LV systolic dysfunction
- Echocardiographic features
  - Contour distinct from endocardial border
  - Often (but not always) more echogenic than underlying myocardium
  - Often (but not always) convex surface
  - Located in region of abnormal wall motion
- Technical suggestions (scanning techniques)
  - Use high-frequency, short-focus transducers
  - Decrease depth of field
  - Move focal zone nearer apex
  - Use low transmit power and gain
  - Addition of nonstandard views
  - Contrast enhancement occasionally helpful
- Pitfalls
  - Near-field and ring-down artifact
  - Prominent trabeculations
  - Papillary muscles
  - False tendons
  - Layered (nonprotruding) thrombi more difficult to appreciate
  - New thrombi are less echogenic
  - May appear and disappear during follow-up
- Potential for embolization
  - Size
  - Protrusion into cavity
  - Mobility

Left atrial thrombus
- Predisposing factors
  - Atrial fibrillation
  - Mitral stenosis
  - Prosthetic mitral valve
  - Left atrial enlargement
  - Low cardiac output
- Association with spontaneous echo contrast
- Location
  - Left atrium
  - Left atrial appendage (poorly imaged with TTE)
  - TEE more sensitive than TTE
- Clinical significance
  - Increased risk of thromboembolism
  - Relative contraindication to balloon mitral valvotomy

Right atrial thrombi
- Predisposing factors
Atrial fibrillation
Right atrium enlargement
Catheters, pacemakers
Clinical significance
  Embolization (pulmonary or paradoxical)
  Thrombi on catheters, pacemakers have potential for infection
Right atrial emboli in transit
Need to distinguish thrombi from:
  Congenital remnants (eustachian valve, Chiari network)
  Microbubbles
  Reverberation artifacts
  Lipomatous hypertrophy of atrial septum
Right ventricular thrombi relatively uncommon

Cardiac Tumors
  Primary
    Benign
      Myxoma
      Papillary fibroelastoma
      Lipoma
      Fibroma
      Hemangioma
      Miscellaneous others
    Malignant
      Sarcomas
        Angiosarcoma
        Rhabdomyosarcoma
        Fibrosarcoma
        Extraskeletal osteosarcoma
      Mesothelioma
      Malignant lymphoma
      Miscellaneous others
  Secondary (metastatic)
    Most often metastasize to visceral pericardium
    Tumors invading right heart via IVC
      Renal cell carcinoma (hypernephroma)
      Hepatocellular
      Uterine tumors
Role of echocardiography for evaluating cardiac tumors
  Detection and characterization
    Morphology
    Location, single, multiple
    Site and nature of attachment
    Infiltration (suggests malignant)
  Differential diagnosis
  Guidance of biopsy, surgery

Miscellaneous Non-Neoplastic Intracardiac Masses
  Mitral annulus calcification
    General
      Degenerative process, increases with age
      Women more than men
    Clinical significance
      Usually of little clinical or functional significance
Conduction disturbance (extension into septum)
Location
Echo features
  Bright, increased echodensity in region of annulus
  May extend into or onto leaflets
  May infiltrate myocardium
  May obscure visualization of thin mitral leaflet

“Atypical” mitral annulus calcification
Terms
  Liquefaction necrosis of mitral annulus calcification
  Sterile, caseous mitral annular “abscess”
Creates suspicious-appearing mass on chest radiograph, echo
  Differential: 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
Cystic masses
  Blood cyst
  Congenital
  Echinococcal cyst

Extracardiac “Masses”
Cysts
  Pericardial
  Bronchogenic
Mediastinal tumors
Aorta
  Tortuous
  Aneurysms

Structures Mistaken for Abnormal Cardiac Mass
Left atrium
  Ridge between left superior pulmonary vein and left atrial appendage
  Atrial suture line after cardiac transplant
  Inverted left atrial appendage
  Atrial-septal aneurysm
  Lipomatous hypertrophy of atrial septum
  Pectinate muscles in left atrial appendage
  Tortuous descending thoracic aorta “compressing” left atrium
Right atrium
  Crista terminalis
  Congenital remnants (eustachian valve, Chiari network)
  Lipomatous hypertrophy of atrial septum
  Trabeculations of right atrial appendage
  Atrial suture line after cardiac transplant
  Catheters, central venous lines, pacemaker wires
Left ventricle
  Papillary muscles
  Anomalous bands (false tendons)
  Prominent muscular trabeculations
  Prominent mitral annulus calcification
Right ventricle
Moderator band
Papillary muscles
Catheter or pacemaker wire
Aortic valve
   Nodules of Arantius
   Lambl’s excrescences
   Aortic cusp imaged *en face* in diastole (TEE)
Mitral valve
   Redundant chordae
   Myxomatous mitral valve tissue
Pericardium
   Epicardial adipose tissue
   Fibrinous debris in chronic pericardial effusion
14. DISEASES OF THE AORTA

Aortic Dissection

Types of dissection
DeBakey: I, II, III
Stanford: A, B
Pathophysiology, Hemodynamics

Echo findings
Visualization of dissection flap
Dilated aorta
Widening of aortic wall (intramural hematoma)
Aortic regurgitation
Pericardial and/or pleural effusion
Compression of left atrium

Goals of imaging
Confirmation or exclusion of diagnosis
Determination of location (i.e., type A or B) and extent of dissection
Presence, severity, and mechanism of aortic regurgitation
Presence of pericardial and/or pleural effusion
Involvement of coronary arteries
Involvement of major branch vessels
Detection of rupture

Less important features
- Localization of intimal tear (entry site)
- Detection of re-entry site(s)
- Flow dynamics with true and false lumens

Transesophageal echocardiography
- Sensitivity, specificity
- Advantages
- Disadvantages
- Superiority over TTE
- Comparison with other imaging modalities
  - Aortography
  - Computerized tomography scan (including fast spiral CT)
  - Magnetic Resonance Imaging

Pitfalls
- Reverberations, catheters
- Mirror-image artifacts
- Spiral flow down descending aorta
- Hemiazygous sheath
- Thoracic aortic aneurysm with mural thrombus
- “Blind spot” (can miss type II dissection)

Intramural hematoma (“atypical” aortic dissection)
Pathogenesis
- Small intimal tears
- Ruptured vasa vasorum
- Penetrating ulcer
- Trauma
Prevalence (10% to 15% of aortic dissections)
Echo findings
- Crescentric or circumferential thickening of aortic wall
- Absence of dissection flap
- Preserved aortic lumen
Clinical significance
Differential diagnosis
- Aortic aneurysm with mural thrombus
- Hemiazygous sheath
- Atherosclerotic thickening of aortic wall

Natural history, fate of false lumen, postoperative complications
- False lumen remains patent approximately 80%
- Post-surgical complications of aortic dissection

Mechanisms of aortic regurgitation

**Thoracic Aortic Aneurysms**
- Definition: True aortic aneurysms are dilatations of the aorta that contain all 3 layers (intima, media, adventitia)
- Pathophysiology: caused by weakening of the media
- Location
  - Ascending aorta (45%)
  - Aortic arch (10%)
  - Descending thoracic aorta (35%)
  - Thoracoabdominal aorta (10%)
- Cause
  - Atherosclerosis
  - Medial degeneration
    - Idiopathic (annuloaortic ectasia)
    - Marfan syndrome
    - Other heritable disorders
      - Associated with bicuspid aortic valve
    - Aortic dissection with dilatation of persisting false lumen
    - Trauma with incomplete aortic rupture
    - Syphilis
    - Myotic (bacterial, fungal, tuberculous aortitis)
    - Noninfectious aortitis (giant-cell, Takayas’ syndrome)
- Echo characteristics
- Role of TEE
- Differentiation from aortic dissection with thrombosed false lumen
- Comparison of TEE with other imaging modalities
  - TEE probably equivalent to Computerized Tomography scan, Magnetic Resonance Imaging, aortography (paucity of data)
  - Each modality has strengths and limitations
- When to operate on ascending aortic aneurysms

**Traumatic Injury of the Aorta**
- Types of injury due to blunt trauma
- Location
  - Usually just distal to origin of left subclavian artery at ligamentum arteriosum (>80%)
  - Occasionally descending thoracic aorta, arch, ascending aorta
- Wide spectrum of extent of injury/pathology
  - Simple contusion
  - Intimal tear
  - Intramural hematoma
  - False aneurysm
  - Frank rupture
  - Major dissection not a feature of aortic trauma (usually no underlying medial disease)
- Lacerations of aortic wall
  - Majority are horizontal
  - May be small, limited, or large, circumferential
Develop from within, extend outward
Intima only
Intima and varying amounts of media
Full thickness of aortic wall
Adventitia is toughest layer

Role of echo (TEE)
Diagnosis
Location and extent of aortic disruption
Associated complications

Diagnostic accuracy of TEE
Limitations of TEE
Advantages of TEE (vs. other imaging modalities)
Comparison of TEE with other imaging modalities (pros and cons)

Aortic Atherosclerosis
TEE
Detection
Grading
Size
Mobility
Ulceration

Clinical relevance
Risk of embolization
Marker for coronary and carotid artery disease, peripheral vascular disease

Management issues
Role of epiaortic imaging in the operating room
Natural history of aortic atherosclerosis

Sinus of Valsalva Aneurysms
Location
Role of echo
Detection (conventional TEE detects 75%)
Delineate location, size, shape of aneurysmal sac
Localize sites of rupture
Identify presence/absence associated abnormalities

Echo features
M-mode
Fluttering of tricuspid valve
Early closure of anterior cusp of aortic valve
Premature opening of pulmonic valve
Right ventricular volume overload

2D echo
Round or fingerlike (windsock) outpouchings
Size and shape may change during cardiac cycle

Doppler
Continuous, high-velocity, turbulent flow
Typically into Right ventricle or right atrium
May be difficult in presence of ventricular septum defect
15. STRESS ECHOCARDIOGRAPHY

**Basic Principles**
- Determinants of myocardial oxygen demand
- Ischemic cascade (sequence of events in ischemia)
- Coronary flow reserve
- Relation between coronary artery anatomy and LV wall segments
- Relation of different ischemic substrates and segmental wall motion/deformation

**Technical and Interpretative Aspects**
- Echo views for evaluation of LV wall motion
- Types of exercise (pros and cons)
  - Treadmill
  - Bicycle
    - Upright
    - Supine
  - Pacing
  - Miscellaneous
    - Handgrip
    - Cold presser
    - Mental stress
- Pharmacologic
  - Dobutamine
  - Dipyridamole
  - Adenosine
  - Atropine
  - Beta-blockers
  - Theofilline
- End points
- Relative contraindications
- Wall motion score index (evaluate before and after stress)
- Interpretation
  - Bayesian analysis
  - Criteria for positive test
  - Interobserver variability
  - Reproducibility
  - Causes of false-positive tests
  - Causes of false-negative tests
- Limitations
- Safety/complications

**Accuracy**
- Sensitivity and specificity
  - In general population
  - In coronary disease population
  - By individual vessels
  - Single vs. multivessel disease
- Comparisons
  - With exercise electrocardiography
  - With nuclear tests
  - Stress echo
    - Treadmill vs. bicycle
    - Exercise vs. pharmacologic
    - Comparison of various pharmacologic agents
**Dypiridamole Stress Echo**

- **Basic principles**
- **Special topics**
  - Role of atropine
  - Hypotension
  - Hyperdynamic response → microvascular disease
- **“Optimal” infusion protocol**
  - Standard: 0.56 mg/Kg over 4 min + 4 min no drug + 0.28 mg/kg over 2 min
  - Accelerated: 0.84 mg/kg in 6 min

**Dobutamine Stress Echo**

- **Basic principles**
- **Special topics**
  - Role of atropine
  - Hypotension
  - LV outflow tract obstruction
- **“Optimal” infusion protocol**
  - Begin with 5 mg for Viability
  - Begin with 10 mg for Ischemia
  - Stages: 3 min vs. 5 min

**Prognostic Role of Stress Echo**

- **Various populations**
  - General referral
  - Known coronary artery disease
  - After acute myocardial infarction
  - After revascularization (thrombolysis, percutaneous transluminal coronary angioplasty (PTCA), coronary artery bypass grafting)

- **Preoperative evaluation before noncardiac surgery**

**Myocardial Viability**

- **Basic principles** (Stunning, Hibernation)
- **Dobutamine stress echo**
- **Interpretation**
  - Biphasic response
  - Improvement at both low and high dose
  - No improvement
  - Comparison with other tests (post-revascularization recovery of function)
    - Positron emission tomography
    - Thallium
    - MRI

**Stress Echo for Hemodynamics and Valve Disease**

- Aortic stenosis
- Mitral stenosis
- Valve regurgitation
- Prosthetic valves
- Pulmonary hypertension
- Hypertrophic cardiomyopathy
- Diastolic function
- Intraventricular gradients
16 TRANSESOPHAGEAL ECHOCARDIOGRAPHY

The Procedure

Laboratory setup
  Equipment, supplies
  Patient preparation

Medication
  Anesthetics (local)
  Sedation (When?, How?)

Technique
  Probe insertion
  Probe manipulation

Relative contraindications
  Pre-existing esophageal pathology
  Esophageal diverticulum
  Esophageal varices
  Recent esophageal surgery
  Active upper gastrointestinal bleed
  Perforated viscus (known or suspected)
  Severe cervical arthritis
  Profound oropharyngeal distortion
  Unwilling or uncooperative patient

Complications
  Arrhythmias
  Respiratory distress, hypoxia
  Transient hypotension or hypertension
  Aspiration
  Laryngospasm, bronchospasm
  Perforation of hypopharynx, esophagus
  Laryngeal nerve damage

Anatomic imaging views

Clinical Indications
17. CONTRAST ECHOCARDIOGRAPHY AND TISSUE HARMONIC IMAGING

Bubble Physics, Pharmacology, Safety

Bubble characteristics
- Size
- Stability (microbubble persistence)
- Radius
- Gas density
- Diffusivity
- Gas composition
- Encapsulation (shell, surface characteristics)
- Saturation concentration

Resonant frequency

Acoustic properties of microbubbles
- Different acoustic impedance than blood
- Intensity of reflections independent of direction of sound source
- High ultrasonic backscatter

External influences on contrast agents
- Ambient pressure
- Acoustic pressure

Safety

Contrast Agents

Ideal contrast agent

Right heart agents (no lung passage)
- Agents (Normal saline, Blood, Albumine)

Characteristics
- Short, variable half-life
- Large, variable size

Clinical indications
- Intracardiac shunt detection
- Intrapulmonary shunt estimation
- Enhance tricuspid regurgitation spectral Doppler tracing

Right and left heart agents (lung passage possible)

Agents

Characteristics
- Stabilizing outer shell
- Nondiffusable gases

Clinical indications
- Enhance LV border delineation
- Enhance spectral Doppler tracings
- Myocardial Perfusion (experimental)

Imaging Instrumentation for Contrast Agents

Imaging modalities for contrast detection

Image mode
- Fundamental mode
- Harmonic imaging
- Color Doppler (fundamental)
- Integrated backscatter
- Power Doppler imaging
- Non-destructive techniques

Capture mode
- Continuous
Triggered (intermittant, gated)
Destruction/fill imaging
Sequential pulse imaging
Analysis mode
   Visual
      Raw image
      Color coding
      Back-ground subtraction
Quantitation
   Densitometry
   Refill Kinetics
   Cyclic variation
Instrumentation issues
   Wide dynamic range
   Narrow transmit spectrum
   Sharp receiver filter
18. REAL-TIME THREE-DIMENSIONAL ECHOCARDIOGRAPHY

Comparison between 2D and 3D

Advantages of 2D echo
- Fast, no post-processing
- Good image quality, high temporal resolution
- Ideally suited for 2D displays
- Easy quantitation of diameters, areas
- 3D reconstruction for experienced examiner no problem

Limitations of 2D echo
- Lack of depth perception
- Complex anatomy incompletely visualized
- Spatial anatomy must be reconstructed mentally
- Communication of mentally reconstructed images is not straightforward
- Requires assumptions of shape for calculations (e.g., LV volume, mass)

Advantages of 3D echo
- Delineate complex orifice shapes
- Delineate complex chamber, mass, structure shape

Limitations of 3D echo
- Limited image quality, limited spatial and temporal resolution
- Full volumes need stitching, sinus rhythm required
- Difficult to display
- Complicated post processing
- Difficult handling of data sets
- Difficult quantitation

Instrumentation:
- Full Matrix Array Transducer (technical aspects)

Display
- Volume Rendering
- Surface Rendering
- Wire Frame
- 2D Tomographic Slices

Quantitation
- Dimensions
- Volumes
- Function
19. TISSUE DOPPLER AND SPECKLE TRACKING

Techniques

Tissue velocity imaging
Principles
- Pulsed-wave tissue velocity
- Color tissue velocity
Postprocessing
- Displacement imaging
- Strain and strain rate
  - Definition
  - Direction of deformation
- Derived Parameters (Phase, Timing of velocity peaks, etc.)

Quantitative analysis

Speckle tracking
Principles
- Physical origin of speckles
- Tracking of speckle motion
Postprocessing
- 2D / 3D application
- Displacement imaging
- Strain and strain rate
- Twist, rotation, torsion
Quantitative analysis

Limitations / Advantages / Disadvantages of both approaches

Clinical applications

Hemodynamic assessment
  - Assessment of filling pressures
Systolic / Diastolic function
Intraventricular dyssynchrony
Myocardial viability
Identification of subclinical myocardial dysfunction (diabetes, obesity, hypertension, heart valve diseases, etc.)
20. SYSTEMIC DISEASES AND OTHER CONDITIONS

Athlete’s Heart
Morphologic changes related to type, intensity, and duration of exercise
Dynamic exercise (e.g., running, skiing, soccer)
  Physiology
    Predominantly volume load
    Substantial increase in cardiac output, heart rate, stroke volume, systolic blood pressure
    Decrease in diastolic blood pressure
  Echo findings
    Increased LV diastolic size
    Increased LV wall thickness, mass ("eccentric" LV hypertrophy)
    Increased right ventricular diastolic dimension
    Increased inferior vena cava dimension
Static (isometric) exercise (e.g., weight lifting, gymnastics, wrestling)
  Physiology
    Predominantly pressure load
    Small increase in cardiac output and heart rate
    No change in stroke volume
    Marked increase in systolic and diastolic blood pressure
  Isometric exercise
  Echo findings
    No significant increase in heart cavity size
    Predominant increase in wall thickness ("concentric" LV hypertrophy)
No changes in LV filling by Doppler (normal transmitral flow velocity)
Important to differentiate athletes’ heart from
  Hypertrophic cardiomyopathy
  Dilated cardiomyopathy
  Arrhythmogenic right ventricular dysplasia

Heart during pregnancy
General (physiology of pregnancy)
  Increased blood volume
  Decreased systemic vascular resistance
  Increased stroke volume and cardiac output
  Increased prevalence of "benign" arrhythmias
Echo findings
  Left ventricular and atrial dilation
  Increased LV stroke volume
  Altered mitral valve coaptation
  Mild tricuspid regurgitation
  Increase in tricuspid regurgitation velocity
Peripartum cardiomyopathy

Systemic Diseases
CARCINOID
  Echo findings (predominantly right-sided valve disease)
    Tricuspid valve thickening, retraction (97%)
    Tricuspid valve may become immobile, fixed in semi-open position
    Pulmonary valve cusps thickened, retracted, immobile (∼50%)0
    Bivalvular involvement common
    Right ventricle and atrium enlargement (∼90%)
Left-sided involvement less common
  Patients with intracardiac shunt (patent foramen ovale) or bronchial tumor
Primary carcinoid in pulmonary bronchus
Mitral valve thickening (5% to 10%)
Moderate to severe Mitral regurgitation (<10%)
Aortic valve thickening (<5%)

Miscellaneous
Carcinoid metastases to myocardium (<5%)
Small pericardial effusions

HEMOCHROMATOSIS
Echo findings
Ventricular wall thickness usually normal
Diastolic dysfunction in early stage
Dilated cardiomyopathy common
Restrictive cardiomyopathy uncommon
Mild valvular regurgitation

SARCOID
Cardiac manifestations
Sudden cardiac death
Arrhythmias
Conduction abnormalities
LV dysfunction and congestive heart failure
Cor pulmonale (second-degree pulmonary sarcoid)
Echo features
Dilated cardiomyopathy (4-chamber enlargement)
Regional wall motion abnormalities
Focal septal thinning with or without aneurysm
Basal posterolateral wall thinning with or without aneurysm
Diastolic dysfunction precedes systolic
If cor pulmonale
Pulmonary hypertension
Right ventricular and atrial enlargement
Papillary muscle dysfunction and mitral regurgitation
Pericardial effusion uncommon (pericarditis)
Restrictive cardiomyopathy if myocardial infiltration

AMYLOID
Echo features
Increased LV/right ventricular wall thickness
Increased myocardial echogenicity (“granular sparkling”)
Restrictive cardiomyopathy
Systolic effects preserved early, poor late
Diastolic dysfunction
E/A > 2
Rapid deceleration time
Valvular thickening and regurgitation (usually mild)
Atrial thrombus
Pericardial effusion

CONNECTIVE TISSUE DISEASES
Rheumatoid arthritis
Echo features
Pericardial (≈ 50% of RA patients)
Effusion (acute pericarditis)
Constrictive pericarditis (<10%)
Effusion-constrictive

Myocardial
- Global LV dysfunction (myocarditis)
- Regional wall motion abnormalities, rare (myocardial infarction from coronary arteritis)
- Diastolic dysfunction
  - Impaired relaxation
  - Not uncommon
- Secondary amyloidosis
- Nodules in myocardium

Valvular/endocardial
- Valve thickening and regurgitation (valvulitis)
- Nodules
- Aortic regurgitation, second-degree aortic enlargement
- Aortic aneurysm, wall thickening; second-degree aortitis (rare)
- Secondary pulmonary hypertension (rare)

SYSTEMIC LUPUS ERYTEMATOSUS

Valvular echo features
- Thickening (especially mitral, aortic)
- Nodularity
- Regurgitation
- Nonbacterial vegetations (Libman-Sacks)
  - Usually <1 cm²
  - Irregular borders
  - No independent motion
- MV prolapse (5% to 10%)

Pericardial echo features
- Effusion (often clinically silent)
- Cardiac tamponade uncommon

Myocardial echo features
- Global LV dysfunction
- Regional wall motion abnormalities
  - Accelerated atherosclerosis
  - Coronary vasculitis
  - Coronary embolism

ANTIPHOSPHOLIPID SYNDROME

Echo features
- Intracardiac, aortic thrombi
- LV systolic dysfunction
  - Regional wall motion abnormalities,
  - Dilated cardiomyopathy
- Valvular regurgitations
- Pulmonary hypertension

SCLERODERMIA

Echo features
- Pulmonary hypertension
- Pericardial
  - Effusion, tamponade, constriction
  - CREST syndrome: symptomatic pericarditis (30%)

Myocardial
- LV hypertrophy with systolic hypertension
- LV dysfunction (up to 75%)
Cardiomyopathy (dilated or restrictive)

MIXED CONNECTIVE TISSUE DISEASE
Cardiac features
  Pericarditis
  Coronary arteritis (rare)
  Myocarditis (rare)
  Pulmonary hypertension second-degree pulmonary disease
  Mitral valve prolapse

ANKYLOSING SPONDYLITIS
Echo features
  Dilatation of aortic annulus and sinus of Valsalva
  Aortic valve thickening
  Aortic regurgitation
  Thickening of aortomitral junction (“subaortic bump”)
  Mitral valve prolapse
  LV systolic dysfunction
  Pericarditis/pericardial effusion (rare)

REITER’S SYNDROME
Echo findings (see ankylosing spondylitis)

MARFAN SYNDROME
Echo findings
  Aortic root dilatation
    Dilated aortic annulus
    Dilated sinuses of Valsalva
    Dilated ascending aorta
    Fusiform ascending aortic aneurysm (annuloaortic ectasia)
    Aortic regurgitation
    Aortic dissection
    Myxomatous mitral valve and mitral valve prolapse
    Mitral regurgitation
    Dilated and calcified mitral annulus
    Giant cell arteritis
    General
      Vasculitis involving large and medium-sized arteries
      Age >50 y
      Increased risk of developing aortic aneurysm
  Echo findings
    Aortic aneurysm and dissection
    Dilatation and thickening of aortic valve and cusps
    LV systolic dysfunction from myocarditis
    Pericardial effusion (pericarditis)

TAKAYASU ARTERITIS
Echo findings
  Dilatation of aorta
  Aortic regurgitation
  Stenosis and occlusion of large vessels

KAWSASKI DISEASE
Cardiovascular
  Vasculitis of coronary vasa vasorum
Leads to coronary artery aneurysms
  Thrombosis
  Stenosis
  Myocardial ischemia, myocardial infarction
Conduction abnormalities
Echo findings
  Coronary artery aneurysms (15% to 25%)
  Small: <4 mm
  Medium: 4 to 8 mm
  Giant: >8 mm
  Pericardial effusion (pericarditis) (30%)
  Myocarditis (common)
  Mitral regurgitation

SYPHILITIC AORTITIS
Echo findings
  Dilated aortic root: aneurysm
  Aortic regurgitation
  Aortic dissection
  Aortopulmonary fistula

HYPEREOSINOPHILIC SYNDROME (LÖFFLER’S)
Echo findings
  LV more often than right ventricular apical cavity obliteration
  Ventricular thrombus
  Cardiomyopathy
    Restrictive
      Biatrial enlargement
      Normal LV and right ventricular size and systolic function
      Restrictive hemodynamics
      Dilated(diffuse myocarditis)
      Thickening/obliteration of inferobasal mitral inflow tract (“entraps,” “plasters down” posterior leaflet)
  Mitral regurgitation (often moderate to severe)
  Variable severity of tricuspid regurgitation
  Pericardial effusion (pericarditis)
  Uncommon
  Constrictive pericarditis
  Asymmetric septal hypertrophy

CHURG-STRAUSS SYNDROME
Cardiac/echo findings
  Pericardial effusion (pericarditis)
  Dilated cardiomyopathy
  Endomyocardial fibrosis

WEGENER’S GRANULOMATOSIS
Echo findings
  LV regional wall motion abnormalities
  Global LV hypokinesis
  Pericardial effusion
  Valvular regurgitation
  LA mass (uncommon)

WHIPPLE’S DISEASE

ENDOCRINE DISEASES
Hyperthyroidism
- Increased stroke volume, cardiac output, and LV mass
- Dilated cardiomyopathy (tachycardia-induced)
- Diastolic dysfunction (impaired relaxation)
- Atrial fibrillation
- Pulmonary hypertension (rare)

Hypothyroidism
- Decreased heart rate and cardiac output
- Prolonged diastolic relaxation
- Dilated cardiomyopathy
- Pericardial effusion (tamponade rare)
- Valvular thickening
- Accelerated atherosclerosis

Pheochromocytoma
- LV systolic dysfunction (catecholamine-induced)
- LV hypertrophy
- Reversible dilatation
- Hypertrophic cardiomyopathy with or without dynamic LV outflow tract obstruction
- Acromegaly

HIV DISEASE (AIDS)
Echo findings
- Pericardial effusion with or without tamponade (up to 40%)
- Infectious (tuberculosis, bacterial, fungal, viral)
- Malignant (lymphoma, Kaposi’s sarcoma, metastatic)
- Non-HIV related cause
- Pericardial constriction
- Dilated cardiomyopathy (up to 50%)
- Myocarditis (HIV, bacterial, fungal, tuberculosis)
- Neoplastic infiltration (lymphoma, Kaposi’s sarcoma)
- Alcohol, nutritional deficiencies
- Cardiac masses
- Neoplasms (Kaposi’s sarcoma, lymphoma)
- Vegetations
  - Marantic endocarditis (common)
  - Infective endocarditis
- Pulmonary hypertension
  - Recurrent pulmonary infections
  - HIV-related interstitial pneumonitis and fibrosis
  - Necrotizing angiitis second-degree drug use
- Thromboembolic events

ERGOT ALKALOIDS, APPETITE SUPPRESSANTS
SYSTEMIC INFECTION/SEPSIS
Echo findings
- Reversible dilated LV with systolic dysfunction (myocardial depression)
- Diastolic dysfunction
- Vegetation
- Pericardial and pleural effusions

HEMATOLOGIC DISORDERS
HEREDITARY HEMORRHAGIC TELANGIECTASIA (OSLER-WEBER-RENDO)
Cardiac involvement
- High cardiac output
- Pulmonary atroventricular malformations (hypoxemia)
- Coronary atroventricular malformations (rare)
Echo findings: contrast echo $\rightarrow$ delayed contrast in LA

CHAGAS DISEASE
Epidemiology
  Rare in North America and Europe
  Endemic in South America
Echo findings
  LV apical aneurysm
  LV posterior wall hypokinesis with minimal involvement of interventricular septum
Role of dobutamine echo
  Unmask chronotropic incompetence
  Limited myocardial contractile reserve
21. PRINCIPLES OF QUALITY ASSESSMENT IN ECHOCARDIOGRAPHY

Principles of Quality Measurements

Individual accreditation
Training
Accreditation
Re-accreditation

Laboratory accreditation

Dimensions of care in echocardiography

Laboratory infrastructure
Baseline standards for equipment
Standards for staff proficiency

Patient selection
Appropriateness of studies
Adequate case mix of pathologies

Study performance
Diagnostic quality of studies
Standardization of performance, storage and reporting echo studies
Recommendations for stress-echo studies
Recommendations for transesophageal-echo studies
Recommendations for contrast-echo studies

Patient safety
Monitoring waiting-list
Clinical prioritization of waiting list
Monitoring complications

Study interpretation
Accuracy
Reproducibility
22. REFERENCES (available at www.escardio.org/communities/EAE/publications/Pages/papers-interest.aspx)


