

**OUTLINE OF CORE CURRICULUM
in
ECHOCARDIOGRAPHY**

American Society of Echocardiography

Core Curriculum Task Force (subcommittee)

**Steven A. Goldstein, MD and Mark Harry, RDCS, RVT
On behalf of the ASE Continuing Medical Education Committee**

CME Committee Members

**Ramon Castello, MD
James V. Chapman, MSc
Ravin Davidoff, MD
Victor G. Davila-Ramon, MD
Steven A. Goldstein, MD
Rebecca Tung Hahn, MD
Mary Jay Harry, RDCS, RVT
Richard Alan Humes, MD
Allan Lawrence Klein, MD
Arthur J. Labovitz, MD
Robert W. McDonald, RCVT, RDCS
Elizabeth F. McIlwain
David J. Mehlman, MD
Thomas Ryan, MD
Kiran B. Sagar, MD
Robert M. Savage, MD
Scott G. Simicich
Marcus F. Stoddard, MD**

CORE CURRICULUM in ECHOCARDIOGRAPHY

Section	Page
1. General Principles of Cardiac Ultrasound	3-11
2. The Echo Exam: Anatomy & Physiology; Technique	12-15
3. Hemodynamics Derived from Echo Doppler	16-31
4. Systolic Function	32-38
5. Diastolic Function	39-44
6. Coronary Artery Disease	45-47
7. Valvular Heart Disease	48-68
8. Diseases of the Myocardium (Nonischemic)	69-76
9. Systemic and Pulmonary Hypertensive Heart Disease	77-78
10. Pericardial Disease	79-81
11. Congenital Heart Disease	82-102
12. Cardiac Masses	103-108
13. Diseases of the Aorta	109-115
14. Stress Echocardiography	116-120
15. Transesophageal Echocardiography	121-122
16. Contrast Echocardiography	123-128
17. Intraoperative Echocardiography	129-136
18. Interventional Echocardiography	137-145
19. Three-dimensional Echocardiography	146
20. Miscellaneous Topics	147-163
Appendix: Sources Used in Compiling Core Curriculum	164-165

Section 1: General Principles of Cardiac Ultrasound

I. Basic Principles of Ultrasound

A. Nature of ultrasound

1. Propagation: compression and rarefaction
2. Differentiation between audible sound and ultrasound

B. Frequency, wavelength, propagation speed in tissues

C. Properties of ultrasound waves

1. Amplitude
2. Power
3. Intensity
4. Pressure

D. Propagation of ultrasound through tissues

1. Average speed of sound in tissues
2. Reflection
 - a. Acoustic impedance
 - b. Reflection and transmission at specular interfaces
 - 1) Interface size, smoothness, and contour
 - 2) Dependence on angle
 - 3) Dependence on acoustic impedance mismatch
 - c. Scattering
 - 1) Scatterer size dependence
 - 2) Frequency dependence (Rayleigh scattering)
3. Refraction
4. Attenuation
 - a. Definition and sources of attenuation
 - b. Variation with frequency
 - c. Effects on images
5. Useful diagnostic frequency range (Tradeoff : penetration vs spatial resolution)

II. Transducers

A. Piezoelectric effect

1. Alteration in shape of crystal when electric charge applied
2. Piezoelectric crystal converts electric energy to sound energy
3. Crystal (element) subjected to rapidly alternating electrical voltage
4. Crystal changes shape rapidly (vibrates)
5. As crystal expands contracts, produces compressions/rarefactions

B. Transducer construction and characteristics

1. Piezoelectric element

- a. Most are artificial ceramics
(eg barium titanate or lead zirconate titanate)
- b. Thickness determines resonant frequency
(thickness inversely proportional to frequency)
- c. Typically, 1 mm thick crystal resonates at 2MHz

2. Damping (due to backing material behind the element)

- a. Shortens the ultrasonic pulse length
- b. Improves range resolution
- c. Also absorbs sound energy directed backward

3. Impedance matching layer(s)

- a. Reduces reflective losses at skin surface
- b. Increases magnitude of acoustic power

C. Sound beam formation

1. Near and far-field (Fresnel and Fraunhofer Zones)
2. Dependence on frequency, transducer size, and bandwidth

D. Focusing method: depth, point of maximal intensity, focal area

1. Methods of focusing (lens, curved element, electronic, mirrors)
2. Focal zone characteristics
 - a. Point of maximum intensity
 - b. Depth of focus
 - c. Focal area

E. Selection of a transducer (tsdr)

1. Size and shape (area or diameter of tsdr face)

a. Size/shape determine the following

- 1) Area encompassed by the sound beam
- 2) Length of near-field
- 3) Angle of divergence in far-field
- 4) Depth at which beam can be focused

b. Advantages of larger size

- 1) Better beam characteristics
- 2) Sharper focus at deeper depth

c. Advantages of small size

- 1) Allows smaller acoustic window
- 2) Easier handling

2. Frequency

a. Resolution (ability to distinguish structures closely related in space as separate)

- 1) Directly related to frequency

b. Penetration

- 1) Inversely related to frequency (increase in frequency increases attenuation)
- 2) In adults, 20 cm or more usually required
- 3) Choice of tsdr frequency also varies with depth of field

3. Focusing characteristics

a. Fixed focal length with acoustic lens (single element)

b. Variable focal length

- 1) Annular array mechanical tsdrs
- 2) Phased array tsdrs

F. Arrays

1. Elements: Linear, phased, annular
2. Beam steering
 - a. Transit time delays
 - b. Receive time delays
3. Beam focusing
 - a. Time delays
 - b. Receive mode
 - c. Multiple transmit foci
4. Mechanical vs phased

III. Imaging Principles of Ultrasound

A. Display modes (advantages and limitations)

1. A-mode
2. B-mode
3. M-mode
4. Two-dimensional

B. Instrumentation

1. Pulsing characteristics
 - a. Pulse repetition frequency
 - b. Duty factor
2. Output power
3. Receiver overall gain
 - a. System gain or coarse gain
 - b. Controls amplitude of all echos within examing plane
4. Receiver swept gain
 - a. Time gain compensation (TGC) selectively amplifies echos at various depths
 - b. Lateral gain compensation (LGC) selectively amplifies echos off central axis of beam

5. Near gain
 - a. Controls amplitude or echos in close proximity of tranducer
 - b. Usually require less amplification
6. Reject
 - a. Selectively filters out all echoes below a certain predetermined amplitude
 - b. Removes echoes that clutter image
7. Damping: adjusts strength of transmitted signal
8. Filter (effect on displayed signal)
9. Signal processing
 - a. Steps in signal processing
 - b. Dynamic range
 - c. Compression
 - d. Pre-processing
 - e. Post-processing

C. Factors influencing 2D-image display and sharpness

1. Analogue and digital signal and scan converters
2. Digital systems: binary system, storage capacity
3. Process of image storage
4. Factors affecting image sharpness and spatial resolution
 - a. Scan converter matrix
 - b. Pixels
 - a. TV lines
 - b. Frequency
 - c. Focal characteristics
5. Display devices
 - a. CRTs and oscilloscopes
 - b. TV monitors

6. Controls effecting signal display

- a. Brightness
- b. Contrast

7. Post processing

- a. Effects on images
- b. Advantages and disadvantages

10. Recording techniques

- a. Hard copy film and paper
- b. Videotape player
- c. Adjustment of contrast/brightness controls

D. Scanning speed limitations

- 1. Pulsing characteristics (pulse repetition frequency)
- 2. Frame rate and time to generate one frame
- 3. Number of lines per frame
- 4. Sector angle (field of view)
- 5. Depth to be imaged
- 6. Temporal resolution

E. Artifacts and pitfalls of imaging

- 1. Reverberations
- 2. Aliasing
- 3. Ghosting
- 4. Mirror images
- 5. Near field clutter
- 6. Range ambiguity
- 7. Refraction
- 8. Shadowing
- 9. Side lobes

IV. Biologic Effects of Ultrasound and Safety

A. Dosimetric quantities

1. Pressure, intensity, power and area
2. Acoustic exposure
 - a. Definition and concepts of prudent use
 - b. Methods of reducing acoustic exposure
 - 1). Factors affecting acoustic exposure
 - 2). Equipment controls

B. Biological effects

1. Cavitation
2. Heat

C. Electrical and mechanical hazards

V. Quality Assurance of Ultrasound Instruments

A. General concepts

1. Need for QA
2. Nature of a QA program

B. Axial resolution

1. Axial resolution
2. Depth calibration accuracy
3. System sensitivity
4. Gray scales display
5. Lesion detection
6. Doppler quality control

VI. Basic Principles of Doppler

A. Physical principles

1. Doppler effect (as related to sampling RBC movement)
2. Doppler equation

$$\Delta f = \frac{2f_t V \cos \theta}{C}$$

Δf = Doppler frequency shift

f_t = transmitted frequency

$\cos \theta$ = angle theta (angle between ultrasound beam and vector of moving object)

C = constant (velocity of sound in tissue, 1560 m/sec)

V = velocity of moving object

3. Range of Doppler shift frequencies
4. Factors influencing magnitude of Doppler shift
 - a. Transducer frequency (transmitted frequency)
 - b. Angle of beam incidence
 - c. Flow velocity
 - d. Frequency conversion

B. Instrumentation

1. Differences between pulsed and continuous wave Doppler (pro/cons)
 - a. Pulsed Doppler
 - (1) Range discrimination and sample volume(s)
 - (2) Aliasing
 - (a) Pulse repetition frequency
 - (b) Nyquist frequency limit
 - (c) Maximum depth
 - (d) Baseline position
 - b. Continuous wave
 - (1) Range ambiguity
 - (2) High velocity measurement capability

2. Spectral analysis and display
 - a. Fast Fourier transformation
 - b. Axis identification
 - c. Gray shade assignments
 - d. Characteristics and information of spectral display
 - e. Spectral broadening and artifacts
 - (1) Influence of sample volume size
 - (2) Pulse width
 - (3) Flow disturbances
 - (4) Electrical interference
3. Color flow imaging
 - a. Sampling methods
 - b. Fundamental variables
 1. Packet size
 2. Line density
 3. Maximum depth
 4. Frame rate
 5. Echo vs color threshold
 - c. Evaluation of frequency content
 - (1) Variance mode
 - (2) Mean frequency
 - d. Color maps
 - e. Artifacts (aliasing, ghosting, reverberation, dropout)
 - f. Display of Doppler information
 - g. Limitations
 - (1) Spatial resolution
 - (2) Color code assignment
 - (3) Maximum velocity
 - (4) Frame rate
4. Tissue imaging principles and information display

Section 2: The Echo Exam

I. Exam

A. Basic imaging principles

1. Tomographic imaging
2. Nomenclature of standard views
3. Image orientation
4. Technical quality

B. Transducer positions, views

1. Parasternal
 - a. Long axis of LV
 - b. Short axis of LV
 - c. RV inflow and outflow views
2. Apical
 - a. Four-chamber view
 - b. Two-chamber view
 - c. Three-chamber view (Long axis)
 - d. Other apical views
3. Suprasternal notch
4. Subxiphoid
5. Other acoustic windows

C. M-Mode echo

1. Aortic valve and left atrium
2. Mitral valve
3. Left ventricle
4. Other M-mode recordings

D. Anatomic basis of 2D-echo

1. Orientation of images (terminology and display)
2. Left ventricular wall segments (as recommended by the American Society of Echo)
3. Coronary artery distribution

E. Principles of echo measurements

1. M-mode
2. 2D-echo

II. Anatomy and Physiology

A. Left ventricle

1. Dimensions, area, volumes
2. LV mass, wall thickness
3. Global systolic function (see section 4)
4. Regional systolic function
5. Diastolic function (see section on Diastolic function)
6. Interdependence of LV and RV

B. Right ventricle

1. Dimensions, area, volumes
2. Global systolic function (see section 4)
3. Echo findings with right ventricular volume overload
4. Echo findings with right ventricular pressure overload
5. Moderator band

C. Left atrium

1. Dimensions, area, volumes
2. LA function

D. Right atrium

E. Ventricular septum

1. Causes of “paradoxical” septal motion

F. Atrial septum

G. LV outflow tract

H. Pulmonary veins

I. Inferior and superior vena cava

J. Great vessels

1. Aorta

- a. Aortic annulus
- b. Sinuses of Valsalva
- c. Sinotubular junction
- d. Ascending aorta
- e. Aortic arch
- f. Descending thoracic aorta
- g. Abdominal aorta

2. Pulmonary artery

- a. Main pulmonary artery
- b. Bifurcation
- c. Right and left pulmonary arteries

K. Coronary sinus

1. Normal imaging
2. Causes of dilatation
3. Differentiation from descending thoracic aorta

L. Coronary arteries

1. Normal imaging
2. Doppler flow patterns
3. Coronary flow reserve

M. Mitral valve apparatus

1. Leaflets
2. Chordae tendinae
3. Annulus
 - a. Normal size
 - b. Variability throughout cardiac cycle
 - c. Nonplanar shape
4. Scallops
5. Papillary muscles

N. Aortic valve

1. Leaflets, commissures, annulus
2. Subvalve, supra valve

O. Tricuspid valve

1. Leaflets (anterior, septal, posterior)
2. Papillary muscles

P. Pulmonic valve

III. Technique

- A. Use of equipment controls
- B. Recognition of technical artifacts
- C. Recognition of setup errors
- D. Use of contrast agents
- E. Provocative maneuvers

IV. Arrhythmias and Conduction Disturbances

- A. Production of wall motion abnormalities
- B. Effect on valve motion
- C. Effect on Doppler flow velocity waveforms

Section 3: Hemodynamics Derived from Echo-Doppler

I. Basic Principles

- A. Laminar vs disturbed (turbulent) flow
- B. Flow velocity profiles

II. Principles of Volume and Flow Measurement

A. Cardiac output

1. Doppler methods

- a. Stroke volume (SV) = Area (CSA) X stroke distance (TVI)
 - 1) CSA: cross-sectional area through which velocity is recorded
 - 2) TVI: time velocity integral or stroke distance
(distance over which blood travels in once cardiac cycle)
- b. Cardiac output (CO) = SV x HR
- c. Assumptions
 - 1) LVOT, RVOT, mitral annulus are circular
 - 2) Annulus area constant during portion of cardiac cycle
 - 3) Area and velocity measured at same site
 - 4) Accurate measurement of LVOT, RVOT, mitral annulus
 - 5) Beam aligned parallel to blood flow
 - 6) Multiple (3 to 5) quality beats measured (5-10 beats in a fib)
 - 7) Modal velocities traced
- d. Potential sites for measurement
 - 1) LVOT
 - 2) RVOT
 - 3) Mitral annulus
 - 4) Tricuspid annulus (much less reliable)

e. Technique for LVOT

1) CSA of LVOT

- a) Parasternal long-axis view, immediately beneath aortic annulus
- b) Mid-systole
- c) Inner edge-to-inner edge
- d) Normal values = 1.8 to 2.2 cm

2) TVI of LVOT

- a) Apical 4-chamber view
- b) Pulsed-wave Doppler
- c) "Inch away" from spectral broadening
- d) Normal values = 16 – 22 cm

f. Technique for mitral annulus

1) CSA at mitral annulus

- a) Apical 4-chamber view
- b) Mid-diastole
- c) Inner edge-to-inner edge
- d) Base of leaflets
- e) Normal values 2.8 to 3.2 cm

2) TVI of mitral annulus

- a) Sample volume at annulus (not tips)
- b) Trace modal velocity
- c) Normal values = 10-14 cm

g. Limitations

- 1) Error in measuring diameter
- 2) Correct placement of sample volume
- 3) Valve regurgitation
- 4) Reproducibility

2. From LV volume (2D-echo)

- a. $SV = \text{difference between LVEDV and LVESV}$
- b. Methods
- c. Pitfalls

B. Shunt ratio

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

a. $Q_p = \text{pulmonary flow} = \text{CSA} \times \text{TVI}$

CSA = area of pulmonic annulus ($A r^2$)

TVI = time velocity integral across pulmonic valve

b. $Q_s = \text{systemic flow} = \text{CSA} \times \text{TVI}$

(usually measured at LVOT)

CSA = area of LVOT ($A r^2$)

TVI = time velocity integral across LVOT

2. Assumptions (same as for cardiac output)

3. Limitations (same as for cardiac output)

C. Regurgitant volume (RV) and regurgitant fraction (RF)

1. RV = volume of blood that regurgitates through incompetent valve

2. RF = fraction of total stroke volume that regurgitates through an incompetent valve

3. $RF = \frac{(\text{forward flow} + \text{regurgitant flow}) - (\text{forward flow})}{(\text{forward flow} + \text{regurgitant flow})}$

4. Values for RF

a. Normal or trivial regurg <20%

b. Mild regurg 20-30%

c. Moderate regurg 30-50%

d. Severe regurg >50%

III. Normal Antegrade Intracardiac Flows

A. Left ventricular outflow

1. Apical 5-chamber outflow

2. Normal values

a. Velocity 0.8 to 1.4 m/sec

b. Aortic TVI 16 to 22 cm

c. Aortic annulus 1.8 to 2.2 cm

B. Right ventricular outflow

1. Parasternal short axis view
2. Sample volume in RVOT or proximal PA
3. Normal values
 - a. Velocity
 - b. Pulmonic TVI
 - c. Pulmonic annulus

C. Left ventricular inflow

1. Apical 4-chamber-view
2. Normal values at mitral leaflet tips (age-dependent)
 - a. E peak early filling velocity 0.8 ± 0.2 m/s
 - b. A peak atrial filling velocity 0.5 ± 0.2 m/s
 - c. A-dur duration of mitral A-wave
 - d. E/A ratio ≤ 1.5
 - e. DT deceleration time 200 ± 40 msec
 - f. IVRT isovolumic relaxation time 70 – 90 msec
 - g. Note: above values represent normals for ages 20-50 years
3. Normal values at mitral annulus (for cardiac output)
 - a. E-velocity
 - b. MV TVI 10 to 13 cm
 - c. Mitral annulus 2.8 to 3.2 cm

D. Pulmonary venous flow

1. Apical 4-chamber view
2. Sample volume 1-2 cm into R-superior vein
3. Normal values
 - a. S₁
 - b. S₂
 - c. D
 - d. S/D
 - e. A amplitude
 - f. A duration

E. Right atrial filling

F. Descending aorta

G. Inferior and superior vena cavae and hepatic vein

H. Coronary arteries (TEE)

1. Left anterior descending

2. Right coronary artery

IV. Assessment of Intracardiac Pressures

A. Bernoulli equation

$$1. P_1 - P_2 = \frac{1}{2}\rho (V_2^2 - V_1^2) + \rho\mu_1 \frac{dv}{dt} ds + R(V)$$

convective flow viscous
accelerate accelerate friction

2. Assumption

- a. Flow acceleration, and viscous friction negligible
- b. Mass density ($\frac{1}{2}\rho$) for normal blood = 4
- c. No energy transfer in system

3. Modified Bernoulli equation

- a. $P_1 - P_2 = 4(V_2^2 - V_1^2)$
- b. $P_1 - P_2 =$ instantaneous pressure gradient
- c. $V_2 =$ accelerated velocity across a stenosis
- d. $V_1 =$ velocity proximal to a stenosis

4. Simplified Bernoulli equation

- a. $P_1 - P_2 = 4V^2$
- b. Assumes V_1 is negligible

5. Pitfalls

- a. Improper beam alignment (large angle θ)
- b. Poorly recorded signals (signal-to-noise ratio)
- c. Failure to detect an eccentric high-velocity jet
- d. Long, tubular stenoses
 - 1) Viscous friction component becomes significant
 - 2) eg. tunnel AS, long coarctation, subpulmonic PS
- e. Changes in viscosity (eg anemia, polycythemia)
- f. V_1 may be significant
 - 1) Esp. with mild stenosis, regurgitation, high output
 - 2) Use $P_1 - P_2 = 4 (V_2^2 - V_1^2)$
 - 3) eg. $V_1 \geq 1.4$ m/sec
- g. Pressure recovery (see Section 3, X)

B. Applications

1. Valvular aortic, pulmonic stenosis
2. Subvalvular aortic, pulmonic stenosis
3. RV or PA systolic pressure
 $4 (\text{TR velocity})^2 + \text{RA pressure}$
4. PA diastolic pressure
 $4 (\text{PR end-diastolic velocity})^2 + \text{RA pressure}$
5. LA pressure
 $\text{Systolic BP} - 4 (\text{MR systolic velocity})^2$
6. RV systolic pressure
 $\text{Systolic BP} - 4 (\text{VSD velocity})^2$

C. Left ventricular diastolic pressure

D. Pulmonary artery pressure/right ventricular pressure

1. M-mode findings
2. Pulmonary acceleration time
3. Tricuspid regurgitant jet method
4. Pulmonic regurgitant jet method
5. Systolic time intervals

E. Right atrial pressure

V. Continuity Equation

A. Basic principle

1. Conservation of mass: Flow proximal to a valve equals flow across the valve

- a. Flow proximal = flow across
- b. $(Area_1) (TVI_1) = (Area_2) (TVI_2)$

2. Rearrangement of continuity equation

$$Area_2 = \frac{(Area_1) (TVI_1)}{(TVI_2)}$$

B. Aortic valve area

1. Technique

a. Measure LVOT diameter (D)

- 1) Parasternal long-axis view
- 2) Immediately beneath aortic valve
- 3) Measure inner border-to-inner border
- 4) Assumes circular geometry

b. Calculate area of LVOT

$$Area_{LVOT} = A(D/2)^2$$

c. Measure LVOT velocity (V_1) and/or TVI_{LVOT}

- 1) Pulsed-wave Doppler
- 2) Apical 5-chamber view
- 3) Immediately beneath aortic valve
 - a) Move sample volume toward aortic valve until flow accelerates
 - b) Then move sample volumes slightly away from valve, toward apex

- 4) Use modal velocity
- d. Measure transvalvular velocity (V_2 , V_{max}) and/or TVI_{AV}
 - 1) CW Doppler
 - 2) Imperative to measure V_{max} from multiple windows (apical, suprasternal, right parasternal, subcostal)
- e. Calculate aortic valve area ($Area_{AV}$)
 - 1) $Area_{AV} = \frac{(Area_{LVOT})(TVI_{LVOT})}{TVI_{AV}}$
 - 2) Not affected by AR
 - 3) TVI or peak velocities give comparable results, but TVI preferred

2. Aortic valve area values

- a. Normal 2.5 to 4.5 cm²
- b. Mild AS 1.2 to 2.4 cm²
- c. Moderate AS 0.8 to 1.1 cm²
- d. Severe AS <0.8cm²

Note: These values somewhat arbitrary; recent ACC/AHA Guidelines on Valvular Heart Disease slightly different

3. Pitfalls

- a. Inaccurate LVOT diameter measurement
 - 1) Oblique image
 - 2) Extensive annular, subvalve calcium
 - 3) “Sigmoid septum”
 - 4) Error is squared
- b. Inaccurate LVOT velocity (V_1)
 - 1) Angle 2
 - 2) Nonlaminar flow if too close to valve (pre-stenotic flow acceleration)
- c. Inaccurate transvalvular velocity (V_2 or V_{max})
 - 1) Underestimation if angle 2 too great

2) Confusion with MR jet

- a) Ejection time (MR begins earlier, extends through IVR period)
- b) Peak velocity (MR > AS)
- c) Aortic clicks, AR jet, mitral inflow

3) Confusion with TR jet (rare)

d. Dynamic subaortic obstruction (obstruction in series)

- 1) Inaccurate LVOT velocity (V_1)
- 2) Use alternate method for measuring AV area

e. Irregular rhythm (eg. atrial fib); average 8-10 beats

f. Low output states (see Section 7, I, B, 6)

- 1) “Falsely” low effective valve area
- 2) Consider inotropic challenge

4. Dimensionless index

- a. Ratio of V_1 to V_2 or V_{LVOT}/V_{max}
- b. Dimensionless index ≤ 0.2 = severe AS
- c. Pitfalls

- 1) Inaccurate V_1 or V_2
- 2) Use of peak velocity vs TVI

C. Mitral valve area

1. Technique

- a. Measure LVOT diameter (see Section 3, V, B, 1, a)
- b. Calculate area of LVOT
- c. Measure LVOT velocity (see Section, 3, V, B, 1, a)
- d. Measure transmitral velocity (TVI_{MV})
 - 1) CW Doppler
 - 2) Multiple windows to optimize velocity and completeness of signal (apical, paraapical, subcostal)
- e. Calculate mitral valve area (MV area)

$$MV \text{ area} = \frac{(\text{Area}_{LVOT}) (TVI_{LVOT})}{TVI_{MV}}$$

2. Pitfalls (similar to AS: see Section, 3, V, B, 3)
 - a. Avoid LVOT if significant AI
 - b. Pulmonic area is alternative

IV. Pressure Half-time Method for Mitral Valve Area

A. Definition of mitral pressure-halftime

1. Time for mitral gradient to fall by half its initial value
2. Time for velocity to fall by $1/\sqrt{2}(0.71 \times V_{\max})$

B. Determinants

1. MV area (orifice area)
2. LV compliance
3. LA compliance
4. LA pressure

C. Technique

1. Optimize CW mitral inflow signal
2. Increase sweep speed to 100 mm/sec
3. Measure peak initial E-wave velocity (V_{\max})
4. Divide V_{\max} by 1.4 ($\sqrt{2}$) to get velocity halftime
5. Draw verticals to baseline at V_{\max} and velocity halftime
6. Measure time interval ($T^{1/2}$)
7. MV area = $220/T^{1/2}$ (220 = empirical constant)

D. Basetime method

1. Optimize CW mitral inflow signal
2. Increase sweep speed to 100 mm/sec
3. Draw vertical to baseline at V_{\max}
4. Extrapolate deceleration of CW signal to baseline (V_0)
5. Measure time interval from V_{\max} to V_0 = deceleration time (DT)
6. Pressure halftime = $(0.29) (DT)$
(0.29 = algebraic constant which converts velocity to gradient)

E. Mitral valve area values

- | | |
|-----------------------------|----------------------------|
| 1. Normal MV area | 4.0 to 6.0 cm ² |
| 2. Mild mitral stenosis | 1.5 to 2.5 cm ² |
| 3. Moderate mitral stenosis | 1.1 to 1.4 cm ² |
| 4. Severe mitral stenosis | ≤1.0 cm ² |

F. Pitfalls

1. Suboptimal Doppler signal
2. Non-linear velocity deceleration slope (non-linear pressure decay)
(use mid-diastolic slope and extrapolate)
3. Acute changes in LA compliance
(eg. immediately post balloon valvuloplasty)
4. Atrial septal defect
5. Significant aortic regurgitation
(shortens $T^{1/2}$, overestimates MV area)

VIII. Proximal Isovelocity Surface Area (PISA) Method for Assessing Valve Regurgitation and Valve Stenosis

A. Definition and principles

1. Regurgitant/stenotic flow converges toward a restrictive orifice in a laminar fashion in ellipsoidal isovelocity surfaces (i.e. multiple concentric “shells”) that approximate hemispheres
2. Principle of conservation of mass applies
 - a. All blood passing through these shells must also pass through the restrictive orifice
 - b. Therefore, flow through any given shell must equal the regurgitant flow ($\text{Flow}_{\text{Regurgitant}} = \text{Flow}_{\text{shell}}$)

B. Assessment of mitral regurgitation

1. $\text{Flow}_{\text{MR}} = \text{Area}_{\text{shell}} \times V_{\text{shell}}$
 $= 2A\mathbf{r}^2 \times V_{\mathbf{r}}$

Flow_{mr} = instantaneous flow rate (cc/sec)

\mathbf{r} = radial distance of the isovelocity shell from orifice (cm)

$V_{\mathbf{r}}$ = flow velocity at radius \mathbf{r} (cm/sec)

2. Effective regurgitant orifice (ERO)

- a. $\text{ERO}_{\text{MR}} = (\text{Flow}_{\text{MR}}) \div V_{\text{MR}}$

ERO_{MR} = effective regurgitant orifice (cm^2)

V_{MR} = peak regurgitant velocity of MR jet (cm/sec)

- b. Average effective area of the regurgitant orifice
- c. Corresponds to severity of regurgitation

- 1) Mild MR <10 mm^2
- 2) Moderate MR 10-25 mm^2
- 3) Severe MR >25 mm^2

3. Regurgitant volume

$$RV_{MR} = ERO_{MR} = ERO_{MR} \times TVI_{MR}$$

$$RV_{MR} = \text{Area of regurgitant orifice} \times \text{TVI regurg signal}$$

$$RV_{MR} = \text{Mitral regurgitant volume (cc)}$$

$$TVI_{MR} = \text{Time velocity integral of MR jet (cm)}$$

4. Assumptions (see Section 7, VII, C,6 d)

5. Advantages

- a. Can be utilized in presence of AR
- b. Quantitation

6. Limitations

- a. Assumption of spherical flow convergence area
- b. Geometry of isovelocity shells change with flow rate and pressure gradient
- c. Flail mitral leaflets may cause a funnel-shaped convergence area (<180°) resulting in overestimation if hemisphere assumed
- d. Inability to accurately measure radius in some pts
- e. High wall filter increases Doppler velocities causing overestimation of flow rate.

C. Assessment of mitral valve area (mitral stenosis)

1. $MV \text{ area} = \text{Flow}_{\text{mitral}} \div V_{\text{peak inflow}}$

a. $\text{Flow}_{\text{mitral}} \text{ (ml/sec)} = 2Ar^2 \times \frac{\text{angle } \nabla}{180^\circ} V_{\text{alias}}$

r = radius of flow convergence region (cm)

angle ∇ = mitral inflow angle (mitral funnel)

V_{alias} = aliasing velocity (cm/sec)

b. V_{peak} = peak CW Doppler velocity of mitral inflow (V_{max})

2. Advantages

- a. Can be utilized in presence of AR
- b. MR does not affect MV area calculation

3. Limitations

- a. Same as with MR (above)
- b. Relatively less well validated than other methods
- c. Higher aliasing velocity (>25 cm/sec) may tend to underestimate MR area

D. Other uses

1. AR
2. ASD and VSD shunt flow
3. Aortic coarctation area

VIII. Valve Resistance (for aortic stenosis)

A. Resistance = $\frac{(\text{Mean pressure gradient}) (\text{SEP}) \times 133}{\text{Stroke volume}}$

1. SEP = systolic ejection period (seconds)
2. Severe AS >255 dynes-sec-cm⁻⁵

B. Clinical utility

1. May be useful in pts with low transvalvular PG
2. Less flow-dependent than aortic valve area
3. Not substantially better than valve area calculation

IX. dP/dt

A. Definition: First derivative of LV pressure rise

B. Utility

1. Indirect measure of myocardial contractility
2. Can be measured noninvasively
3. Relatively afterload independent

C. Assumptions

1. CW Doppler velocity of MR reflects instantaneous PG between LV/LA
2. LA is compliant (LA pressure stable during pre-ejection period)

D. Technique

1. Optimize CW MR jet with clear initial slope
2. Use high filter setting
3. Decrease velocity range to maximize envelope from baseline to 4 m/sec
4. Increase sweep speed to 100 mm/sec
5. Draw horizontal lines at 1 m/sec and 3 m/sec
6. Draw vertical line from intercept of MR jet at 1 m/sec and 3 m/sec
7. Measure time (dt) between these 2 points (msec)
8. $dP/dt = 32,000/dt$ (mmHg/sec)

E. dP/dt values

- | | |
|---------------|--------------------|
| 1. Normal | >1200 mmHg/sec |
| 2. Borderline | 1000-1200 mmHg/sec |
| 3. Abnormal | <1000 mmHg/sec |

F. Pitfalls of dP/dt

1. Poor alignment of CW cursor with MR jet (underestimates)
2. Acute MR (LA noncompliant, LA pressure rises with MR)
3. Preload dependent

X. Pressure Recovery

A. Basic principles

1. As flow goes through a stenotic orifice, it narrows then expands
 - a. Pressure along flowstream is lowest at vena contracta
 - b. As post-stenotic jet expands beyond the vena contracta:
 - 1) Flowstream reattaches to vessel wall
 - 2) Velocity and kinetic energy decrease
 - 3) Potential energy (**pressure**) rises

2. Doppler: measures max PG

Between highest pressure (prox to stenotic orifice)
and lowest pressure at vena contracta

3. Cath: measures lower PG
 - a. Between highest pressure (prox to stenotic orifice)
 - b. and recovered pressure, several cm above stenotic orifice

B. Clinical significance

1. Cath gradient $<$ Doppler gradient (native valve)
2. Cath gradient \ll Doppler gradient in certain prosthetic valves
 - a. Especially small size bileaflet mechanical valves (19,21)
 - b. Also, small size ball-cage valves
 - c. To lesser extent, small stented, bioprosthetic valves

Section 4: Systolic Function

I. Determinants of LV Performance

A. Contractility (inotropic state of myocardium)

1. Well-defined concept *in vitro*
(velocity of shortening of isolated muscle strip)
2. More difficult to measure clinically
(dependent on preload and afterload)
3. End-systolic elastance of ventricle
 - a. *Approximately* independent of preload and afterload
 - b. $E_{ES} = P_{ES} / (V_{ES} - V_o)$
 P_{ES} = end-systolic pressure
 V_{ES} = end-systolic volume
 V_o = dead space term
 - c. Determined (strictly) by evaluating ventricular P-V loops for different preloads or afterloads

B. Preload

1. **Fiber length at onset of contraction**
2. Frank-Starling relationship
 - a. Myocardial work increases as resting length of myocardial fiber increases
 - b. Stroke volume increases as end-diastolic volume increases
3. End-diastolic volume
(extrapolation of *in vitro* studies of isolated muscle strips)
4. Echo assessment of preload (see Section 3, II-A)
 - a. LVEDV
 - b. LV diastolic pressure

C. Afterload

1. Counter force to contraction which halts shortening when it equals the force generated of the myocardium
2. Determinants
 - a. Ventricular volume and pressure
 - b. Arterial resistance
 - c. Aortic impedance
 - d. Mass of blood in aorta
 - e. Viscosity of blood

II. Global LV Systolic Function

A. Measurements

1. Ejection phase indices

a. Indices

- 1) Ejection fraction (EF)
- 2) Fractional shortening (FS)
- 3) Velocity of circumferential fiber shortening (Vcf)
- 4) Cardiac output (CO) and stroke volume (SV)

b. Potential limitations

- 1) Requires high-quality images (endocardial definition)
- 2) Some methods require no major shape distortion
- 3) Beam orientation (avoid oblique measurements)
- 4) Limited frame rate
- 5) Foreshortening of long axis of LV
- 6) Influence of loading conditions

2. Non-ejection phase indices

a. Systolic time intervals

b. dP/dt

c. Acceleration time

d. Pressure–volume analysis

- 1) Pressure-volume loops
- 2) End-systolic stress-dimension relations
- 3) End-systolic stress-volume relations

e. Stress-shortening relations

- 1) Avoids afterload dependency
- 2) Difficult to determine

3. Indirect methods (simple confirmatory methods)

- a. Mitral E-point septal separation (≥ 8 mm \approx EF $< 50\%$)
- b. Aortic root motion (normally > 14 mm during systole)
- c. Descent of cardiac base (normally moves 10-15 mm)
- d. Sphericity of left ventricle
- e. TVI of LVOT and pulmonary artery velocities

B. What to measure

1. Ejection fraction (EF)

- a. Ratio of stroke volume and end-diastolic volume
- b. Most commonly used clinical index of LV function
- c. Dependent on:
 - 1) Preload
 - 2) Afterload
 - 3) Heart rate
- d. Normal EF = $61 \pm 10\%$
- e. Reproducibility $\pm 10\%$ (95% CI)
- f. Visual estimation
 - 1) Generally valid by experienced echocardiographer
 - 2) Interobserver variability
- g. Quantitation
 - 1) Measurement of LV diameters
 - a) Minor axis diameters
 - b) Assumption: LV is prolate ellipsoid
 - c) Limited with RWMA, abnormal LV shape
 - d) Does not incorporate changes in long-axis length
 - 2) Measurement of LV volumes (see Section 4 II-C)
 - a) Area – length method
 - b) Modified Simpson's rule
- h. Examples of situations where limited (markedly altered loading conditions)
 - 1) MR – EF often normal in presence of intrinsic myocardial dysfunction
 - 2) AS – EF may be decreased in presence of normal myocardial function
 - 3) Severe anemia
 - 4) Hemodialysis patients

2. Fractional shortening (% FS)

- a. Simple M-mode echo technique
- b. $EDD - ESD/EDD$
- c. Like EF, is load dependent
- d. Poor in asymmetric ventricles
- e. Normal $\geq 29\% \pm 5\%$

3. Velocity of circumferential fiber shortening (Vcf)

a.
$$Vcf = \frac{LVIDd - LVIDs}{LVIDd \times ET}$$

LVIDd = LV internal dimension (diastole)

LVIDs = LV internal dimension (systole)

ET = Ejection time

- b. Normal Vcf > 1.1 circumferences per second

4. End-systolic volume (ESV)

- a. Most reproducible volume measurement
- b. Relatively insensitive to cardiac loading
- c. Powerful predictor of cardiac events
- d. Normal values

Males	34 ml
	58 ml (90% upper confidence bound of 95 th %)
	18 ml/m ²
Females	29 ml
	50 ml (90% upper confidence bound of 95 th %)
	18 ml/m ²

- e. Reproducibility $\pm 15\%$ (95% CI)

5. End-diastolic volume (EDV)

- a. Endocardium more difficult to image at end-diastole
- b. More variable than ESV

c. Normal values

Males	111 ml 156 ml (90% upper confidence bound of 95%) 58 ml/m ²
Females	80 ml 105 ml (90% upper confidence bound of 95%) 50 ml/m ²

d. Reproducibility ± 25% (95% CI)

6. LV mass

a. Methods

1) M-mode methods

a) Penn formula:

$$\text{LVM} = 1.04 \times [(\text{Dd} + \text{S} + \text{PW})^3]$$

Dd = LV end-diastolic diameter

S = Septal thickness

PW = Posterior wall thickness

1.04 = Specific gravity of LV muscle

b) Am Society Echo equation:

$$\text{LVM} = 0.80 \times 1.04 \times [(\text{Dd} + \text{S} + \text{PW})^3 - (\text{Dd})^3] + 0.6$$

2) 2D-methods

a) Area-length (instrument analysis package)

b) Truncated ellipsoid (instrument analysis package)

c) Longitudinal method

$$\text{LVM} = 0.687 \times [(\text{Dd} + \text{S} + \text{PW})^2 \times (\text{L} + \text{PW})] \times [\text{Dd}^2 \times \text{L}]$$

Dd, S, PW from parasternal SAX (pap level)

L from apical 4 or 2 chamber (whichever longer)

b. Limited by relatively wide standard deviations

c. Requires change >35 gm to reach 95% CI

d. Normal values:

Males	235 gm 183 gm 90% upper confidence bound of 95%) 71 gm/m ²
Females	99 gm 141 gm 90% upper confidence bound of 95 th % 62 gm/m ²

C. Methods for LV volumes (ESV, EDV)

1. Biplane method of discs (modified Simpson's rule)
 - a. Represents cavity as stack of coins or discs
 - b. Computer automatically sums individual volumes of each disc
 - c. Most closely predicts angiographic volume
 - d. Depends on LV nearly equal length in 2 orthogonal views
2. Single plane area-length
 - a. $V = 0.85 (\text{Area})^2/L$
 - b. Slightly less accurate (suitable if LV symmetric)
3. "Quick" method (regression equation)
 $EDV = (3.42 \times D \times L) - 6.44$

D. Technical considerations

1. Image maximization
(echo "slice" must pass through largest dimension of chamber)
 - a. Center image so neither anterior nor posteriorly canted
 - 1) Both AV valves imaged
 - 2) Avoid aorta (anterior) and coronary sinus (posterior)
2. Selection of precise time in cardiac cycle for measurements
 - a. End-diastolic frame
 - 1) Largest LV cavity
 - 2) Just before MV closure
 - b. End-systolic frame
 - 1) Smallest LV cavity
 - 2) Just before MV opening
3. Potential Problems
 - a. Endocardial dropout (esp. apical views)
 - b. Foreshortening of LV cavity (will overestimate EF)
 - c. Regional wall motion abnormalities
 - d. Translation
 - e. Discoordination of contraction (eg. LBBB, pacemaker)

4. New technologies to improve endocardial delineation
 - a. Harmonic images
 - b. Newer contrast agents

E. Clinical significance

1. Prognosis in CAD, acute MI
2. Prognosis in DCM
 - a. EF
 - b. Relative wall thickness
 - c. Volume/mass ratio
3. Therapeutic implications (ACE-inhibitors)
4. Timing of surgery in volume overload states (MR, AI)
 - a. EF
 - b. EDV, ESV
 - c. EDV/mass
 - d. Systolic stress-shortening relations
 - e. End systolic P-V relations
1. Timing of surgery in pressure overload states (AS)
 - a. EF less important
 - b. High relative wall thickness (increased risk)
2. Hypertension
 - a. LV mass predicts morbidity, mortality
 - b. LV wall thickness/cavity dimension predicts morbidity/mortality

A. Associated Findings

1. Thrombi
2. Changes in appearance of involved myocardium
3. Valve dysfunction

III. Regional systolic function

IV. Interdependence of LV and RV

V. Global RV systolic function

Section 5: Diastolic Function

A. Basic Principles

A. Phases of diastole

1. Isovolumic relaxation
2. Early rapid diastolic filling
3. Diastasis
4. Late diastolic filling due to atrial contraction

B. Parameters of diastolic function

1. Relaxation

- a. Isovolumic relaxation time
- b. Maximum rate of pressure decline (peak negative dP/dt)
- c. Time constant of relaxation (τ)

2. Compliance

- a. dP/dV
- b. Chamber stiffness constant (k)

3. Ventricular diastolic pressures
4. Ventricular diastolic filling (volume) curves

II. Echo-Doppler Approach to LV Diastolic Function

A. Digitized M-mode

B. 2D-echo

1. Chamber dimensions
2. Wall thickness

C. Doppler measures of LV filling

1. Mitral E-wave velocity
2. Mitral A-wave velocity
3. E/A ratio
4. Mitral A-wave duration
5. IVRT
6. Deceleration time, deceleration slope
7. Time from mitral valve opening to E-velocity
8. Pulmonary venous flow
 - a. S/D ratio (normal >1; age related)
 - b. AR-wave amplitude
 - c. AR-wave duration minus mitral A duration
 - d. Limitations
 - 1) Technically difficult to obtain clear AR velocity tracings
 - 2) May be reduced in some patients with “pseudonormal” and restrictive filling (presumably due to atrial mechanical failure)

D. Normal and abnormal Doppler patterns

1. Normal
 - a. Mitral E slightly >A
 - b. DT 200 ± 40 msec
 - c. IVRT 70 – 100 msec
2. Abnormal relaxation
 - a. Indicates rate of ventricular relaxation prolonged
 - b. Doppler features
 - 1) $E \ll A$
 - 2) DT >240 msec
 - 3) IVRT >100 msec
 - c. Significance
 - 1) Earliest manifestation of diastolic dysfunction
 - 2) No increase in resting mean LA pressures
 - 3) May cause problem with high HR and/or loss atrial contraction
 - 4) Seen in HTN, CAD, HCM, early DCM, elderly

3. Pseudonormal

a. Underlying abnormality of relaxation, but normalization due to increased LA pressure

b. Doppler features (looks like normal pattern)

- 1) E slightly >A
- 2) DT = 160 – 240 msec

c. Significance

- 1) Underlying abnormality of relaxation, but ...
- 2) Normalization of mitral flow velocity curve due to increased LA pressure
- 3) Can be mistaken for normal

d. Distinguishing pseudonormal from normal/LV filling

- 1) Normal LA size suggests normal LV filling
- 2) IVRT <70 msec suggests pseudonormal pattern
- 3) Pulmonary venous A-dur >mitral A-dur (>30 msec) suggests increased LA pressure → pseudonormalization
- 4) Preload reduction unmasks relaxation abnormality

- a) Valsalva
- b) NTG
- c) Diuresis

- 5) Color M-mode
- 6) DTI

4. Restriction to filling

a. Doppler features

- 1) E >>A (E/A >1.5)
- 2) DT <160 msec
- 3) IVRT <70 msec

b. Significance

- 1) Late stage of diastolic dysfunction
- 2) Severe decrease in LV compliance
- 3) Rapid filling of LV in early diastole
- 4) Elevation of mean LA pressures at rest
- 5) Seen in DCM, RCM, ischemic CM
- 6) May reverse with optimal mgt of CHF
(DT may increase over time)

5. Irreversible restrictive

- a. As above
- b. Doesn't change with optimal mgt of CHF
- c. Poor prognosis

E. Factors that affect LV filling pattern (Determinants of filling indices)

1. Technical

- a. Sample volume location
- b. Doppler modality (PW vs CW)
- c. Intercept angle

2. Normal variations

- a. Respiration
- b. Heart rate
- c. PR interval
- d. Age

3. Physiologic

- a. Preload
- b. Afterload
- c. Exercise
- d. Valsalva
- e. LV systolic function and end-systolic volume
- f. LV minimal pressure
- g. Atrial contractile function
- h. Diastolic function

- a) Relaxation
- b) Compliance/stiffness

4. Cardiac rhythm

F. Clinical applications (conditions associated with diastolic dysfunction)

1. Hypertension, LVH
2. Diabetes
3. Dilated cardiomyopathy
4. Hypertrophic cardiomyopathy
5. Restrictive cardiomyopathy
6. Ischemic heart disease
7. RVVO and RVPO
8. Valvular heart disease (eg aortic stenosis)
9. Pericardial disease
10. Transplant rejection

A. Estimation of LV filling pressure (PCW, LVEDP, LA)

1. Correlation best in dilated cardiomyopathy
2. Poor correlation in HCM
3. DTI : E/Ea
4. Color M-mode: E/VP

B. Progression for normal to severe diastolic dysfunction

1. Progressive increased preload compensation
2. Parabolic or U-shaped pattern

C. Role in prognosis (DT important predictor)

I. Newer methods of evaluating diastolic function

1. Mitral E and A-wave transmission inside LV
2. Color M-mode (LV flow propagation)
3. Tissue Doppler Imaging (Tissue Doppler echocardiography)
4. Color kinesis

III. Right Ventricular Diastolic Function

A. Factors that may affect RV filling

1. Technical
 - a. Sample volume location
 - b. Intercept angle

2. Normal variation
 - a. Respiration
 - b. Heart rate
 - c. PR interval
 - d. Age

B. Doppler measures of RV filling

1. Tricuspid E-wave
2. Tricuspid A-wave
3. Deceleration time
4. IVC size and degree of inspiratory collapse
5. Hepatic vein and SVC flow
 - a. S/D ratio
 - b. AR and VR amplitude

6. Effect of respiration

C. Abnormal RV Doppler patterns

1. Abnormal relaxation
2. Pseudonormal
3. Restrictive

D. Clinical Applications

1. Pericardial disease
 - a. Tamponade
 - b. Restriction
2. Restrictive cardiomyopathy
3. Cor pulmonale

E. Estimation of RV filling pressures

1. IVC plethora
2. Hepatic vein systolic fraction
3. $DTI = E/Ea$

F. Newer methods

1. Tissue Doppler imaging
2. Color kinesis

Section 6: Coronary Artery Disease

I. Coronary Artery Anatomy and Function

A. Flow

1. Coronary artery distribution
2. Relationship between coronary arteries and corresponding myocardial territories
3. Flow measurements and limitations
4. Coronary flow reserve
5. Myocardial perfusion using contrast echo

B. Anatomy

1. Normal coronary imaging (parasternal short axis)
 - a. L-main at 4:00
 - b. RCA at 11:00
2. Coronary atherosclerosis

II. Coronary Artery Abnormalities

A. Anomalous origin or course (see Section 11-XI-A)

B. Coronary aneurysms

1. Kawasaki's disease
2. Atherosclerotic aneurysms
3. Polyarteritis
4. Syphilis
5. Infection
6. Trauma

C. Coronary fistulae

1. Rare congenital anomaly
2. Estimated population incidence is 0.002%
3. Involved cor art empties into cardiac chamber or PA
4. RCA 50%; LCA 45%; both 5%
5. RV 40%; PA 25%; LA 15%; cor sinus 5%; LV 5%
6. Continuous murmur

7. Echo-Doppler findings

- a. Dilated coronary artery (>3 mm)
- b. Chamber enlargement
- c. Color Doppler may detect shunt flow at receiving chambers

III. Myocardial Ischemia

A. Basic principles

1. Sequence of events in ischemia
2. Relationship of wall motion and wall thickening to coronary artery perfusion

B. Detection of ischemic muscle

1. Method

- a. Endocardial motion
- b. Thickening/thinning

2. Limitations, technical aspects

3. Confounding factors

- a. Conduction or pacing abnormalities
- b. Translational motion
- c. Loading conditions
- d. Altered imaging planes

C. Stress echocardiography (see section on Stress Echo)

D. Diastolic function changes

IV. Myocardial Infarction

A. Detection and location of MI

1. Regional wall motion abnormality
2. Relationship between transmural and regional function
3. Non-CAD causes for chest pain

- a. Aortic stenosis
- b. Hypertrophic cardiomyopathy
- c. Pericarditis (with effusion)
- d. Aortic dissection

- B. Quantitation of MI
 - (prognostic value: guide therapy)
- C. Limitations and alternate approaches
- D. Complications and associated findings
 - 1. Free wall rupture
 - 2. Ventricular septal rupture
 - 3. Aneurysm, pseudoaneurysm
 - 4. Papillary muscle rupture
 - 5. Right ventricular infarction
 - 6. LV thrombus
 - 7. Infarct expansion and extension
- E. Follow-up transthoracic echo
 - 1. Remodeling (infarct expansion; global dilatation)
 - 2. Recovery of function
 - 3. LV thrombus

Section 7: Valvular Heart Disease

I. Valvular Aortic Stenosis

A. Etiology and imaging of the aortic valve (aortic valve morphology)

1. Congenital
 - a. Bicuspid aortic valve
 - b. Unicuspid aortic valve
2. Rheumatic
3. Degenerative (calcific)

B. Quantitation

1. Pressure gradients (see also Section 3, V, A)

a. Measure maximal velocity from at least 2 sites

- 1) Apical 5-chamber
- 2) Suprasternal
- 3) Right parasternal
- 4) Subxiphoid

b. Assessment of gradient unreliable in subaortic stenosis

- 1) Bernoulli equation invalid with obstruction in series in close proximity
- 2) Cannot measure V_1 (LVOT velocity)
- 3) Alternative method:

$$\text{MR velocity } (4V^2) + \text{LA Pr } (\approx 15) = \text{LV}_{\text{systolic Pr}}$$
$$\text{LV}_{\text{syst Pr}} - \text{LVOT}_{\text{gradient}} - \text{Syst BP} = \text{AV}_{\text{gradient}}$$

2. Continuity equation valve area (see Section 3, V, B)

- a. Technical considerations
- b. Limitations, pitfalls

3. Dimensionless index (velocity ratio)

4. Planimetry of stenotic orifice using TEE
 - a. TTE inaccurate
 - b. TEE appears to work
 - 1) Technique
 - a) Obtain optimal longitudinal view of aortic root/valve (often $\approx 120^\circ$)
 - b) Subtract 90° to obtain optimal cross-sectional view
 - 2) Limitations
 - a) Failure to image smallest orifice (esp bicuspid valve – doming)
 - b) Shadowing and blooming artifacts due to calcium
5. Valve resistance (see Section 3, VIII)
6. Low-gradient aortic stenosis
 - a. Baseline hemodynamics
 - 1) Low cardiac output
 - 2) Gradient < 30 mmHg
 - 3) Small effective orifice area (< 1.0 cm²)
 - b. Two possible explanations
 - 1) True anatomic stenosis (true AS)
 - 2) Primary contractile dysfunction (mild or moderate AS)
 - c. May distinguish using exercise or pharmacologic stress echo
 - 1) True AS \rightarrow no change or minimal increase valve area
 - 2) Mild/mod AS \rightarrow valve area increases

C. Response of the LV to valvular AS

1. Quantify LV hypertrophy
2. Measure LV dimensions
3. Assess LV function

D. Measure aortic root (at 3 levels if dilated)

1. Annulus, sinuses, sinotubular junction
2. Sizing for homograft, Ross procedure, stentless valves

II. Mitral Stenosis

A. Etiology and diagnostic imaging

1. Rheumatic
2. Mitral annular calcification and calcific MS
3. Congenital
4. Miscellaneous
 - a. Myxoma, other tumors
 - b. Cor triatriatum

B. Echo findings

1. Leaflet thickening, especially tips
2. Immobility of posterior leaflet
3. Commissural fusion
4. “Doming” of anterior mitral leaflet (diastole)
5. Calcification (leaflets, commissures, chordae, annulus, pap muscles)
6. Chordal thickening, fusion (may obliterate secondary orifices)
7. Funnel-shaped mitral apparatus
8. Decreased mitral opening

C. Quantitation

1. Pressure gradients
 - a. Peak (initial gradient)
 - b. Mean gradient
 - 1) More accurate than cath using PCWP and LVP
 - 2) Highly dependent on HR (record HR at which measured)
 - 3) Depends on cardiac output
2. MV area
 - a. Planimetry
 - b. Pressure half-time method
 - c. Continuity equation
 - d. PISA method
 - e. Technical considerations and pitfalls of each method

D. Coexisting Lesions

1. Left atrial enlargement
2. Pulmonary hypertension
3. MR, AS, AI, TS, TR
4. Left ventricular response
5. Right ventricular response
6. Thrombi in LA and LA-appendage

E. Role of hemodynamic stress testing (exercise or dobutamine)

1. When to consider
 - a. Discrepancy between symptoms and resting hemodynamics
 - b. Pts. with sedentary lifestyle (evaluate ex. tolerance, HR, BP)
2. Significant findings
 - a. Rise in mean transmitral gradient (to >15 mmHg)
 - b. Rise in PA systolic pressure (to >60 mmHg)

F. Percutaneous Balloon Mitral Valvotomy – Role of Echo

1. Patient selection (suitability for PBMV)
 - a. Mitral valve score (based on morphology of MV apparatus)
 - 1) Leaflet mobility
 - 2) Leaflet thickness
 - 3) Leaflet calcification
 - 4) Subvalvular thickening, fusion
 - b. Mitral regurgitation $\leq 2+$
 - c. Absence of thrombi in left atrium
 - d. Minimal or no commissural calcium
 - e. Other features
 - 1) Anatomy of atrial septum
 - 2) Type of mitral “funnel”

2. Echo guidance *during* valvotomy
 - a. Guidance of transseptal puncture
 - b. Guidance of balloon positioning
 - c. Immediate assessment of results
 - 1) Mean gradient
 - 2) Mitral valve area (P ½ T method)
 - 3) Separation of leaflets
 - 4) Pulmonary venous flow
 - d. Detection of complications
 1. Mitral regurgitation (degree, mechanism)
 2. Tamponade (LV or LA perforation)
 3. Atrial septal defect

G. Recommendations for TEE

1. Assess for LA thrombi in pts. being considered for PBMV or cardioversion
2. Evaluate MV morphology and/or hemodynamics when TTE suboptimal

III. Tricuspid Stenosis

A. Etiology

1. Rheumatic
2. Congenital
3. Carcinoid
4. Fabry's disease
5. Previous methysergide therapy

B. Echo-Doppler

1. Tricuspid valve structure and motion
2. TV diastolic gradient
3. Other cardiac abnormalities

IV. Pulmonic Stenosis (see section 11, IV, E)

V. Basic Principles of Valve Regurgitation

A. Fluid dynamics of valvular regurgitation

1. Regurgitant orifice (size, shape)
2. Proximal flow acceleration
 - a. Converging streamlines on the high-pressure side
 - b. Proximal isovelocity surface area (PISA)
 - c. Can be used for quantitation
 - 1) Peak flow velocity
 - 2) Regurgitant orifice area
3. Vena contracta
 - a. High velocity laminar flow through narrow regurgitant orifice
 - b. Narrowest part of regurgitant jet
 - c. Directly proportional to anatomic orifice size
 - d. Useful measurement for both MR and AR
4. Flow disturbance into low-pressure receiving chamber
 - a. Composition
 - 1) Area of turbulence (jet area)
 - 2) Entrainment of surrounding RBCs
 - b. Jet area
 - 1) Useful for semi-quantitation of regurgitation
 - 2) Caution: not equivalent to regurgitant volume
5. Increased volume flow across valve
 - a. Increased antegrade velocity
 - b. Volume flow at 2 sites
 - 1) Can determine regurgitant volume, fraction
 - 2) Pitfalls/limitations
6. Conservation of momentum
 - a. Regurgitant orifice area

- b. Momentum

- B. Factors that affect regurgitant jet size and shape

- 1. Physiologic

- a. Regurgitant volume
 - b. Driving pressure
 - c. Size and shape of regurgitant orifice
 - d. Receiving chamber constraint
 - e. Wall impingement
 - f. Timing relative to cardiac cycle
 - g. Influence of coexisting jets or flowstreams

- 2. Technical

- a. Gain
 - b. Pulse repetition frequency
 - c. Transducer frequency
 - d. Frame rate
 - e. Image plane
 - f. Depth

- C. Detection of valve regurgitation

- 1. Indirect methods

- a. Valve anatomy
 - b. Chamber dilatation and function

- 2. Direct methods

- a. Pulsed Doppler
 - b. Continuous wave Doppler
 - c. Color flow imaging

- D. Valvular regurgitation in normal individuals

- E. Quantitation of regurgitation severity

- 1. Semiquantitation

- a. Flow mapping (pulsed or color)
 - b. CW Doppler signal intensity
 - c. Distal flow reversals

2. Quantitative
 - a. Volume flow at 2 sites
 - b. Descending aorta flow reversal (ratio of forward to reverse flow)
 - c. Proximal isovelocity surface area
 - d. Jet momentum

VI. Aortic Regurgitation

A. Imaging of the valve apparatus (etiology, mechanism)

1. Leaflet abnormalities
 - a. Congenital abnormalities (unicuspid, bicuspid)
 - b. Degenerative valve disease (fibrosis/sclerosis)
 - c. Rheumatic valve disease
 - d. Endocarditis
 - e. Miscellaneous other entities
2. Aortic root abnormalities
 - a. Hypertension
 - b. Bicuspid valve
 - c. Annuloaortic ectasia
 - d. Marfan's syndrome
 - e. Aortic dissection
 - f. Miscellaneous other entities

B. Indirect signs of aortic regurgitation

1. Left ventricular dilatation and sphericity
2. Left ventricular hyperkinesis (until late)
3. Increased E-point septal separation
4. High frequency fluttering of anterior mitral leaflet (AMVL) does not correlate with severity
5. "Reverse doming" of AMVL (posteriorly displaced anterior mitral leaflet)
6. Jet lesion on septum or AMVL
7. Premature aortic valve opening
8. Increased LVOT velocity
9. Diastolic mitral regurgitation

C. Severity of aortic regurgitation

1. Flow mapping (pulsed or color)
 - a. Quick, easy, but semiquantitative
 - b. Jet length, height, area, ratio jet height/LVOT width
 - c. Dependent on physiologic and technical factors (see Section 7, V,B)
2. CW Doppler signal intensity
 - a. Intense CW signal indicates large regurgitant volume
 - b. Limitations
 - 1) Semiquantitative
 - 2) Technical factors
 - a) Somewhat gain dependent
 - b) Poor alignment of jet and imaging beam (esp eccentric and curvilinear jets)
 - c) Other factors may affect signal intensity
3. Shape of CW Doppler time-velocity curve
 - a. Rapid decline indicates severe AR
 - b. Assumptions
 - 1) Aortic pressure falls rapidly
 - 2) LV filling pressure rises rapidly
 - c. Technical considerations
 - 1) Obtain from apical 5-chamber view (alternately 3-chamber view)
 - 2) Clear, continuous envelope throughout diastole
 - 3) Not influenced by beam angulation
 - d. Pitfalls
 - 1) Poor quality tracing
 - 2) Affected by other factors
 - a) Compliance of LV, aorta
 - b) Stroke volume
 - c) Afterload

- d) Systolic vascular resistance
4. Holodiastolic flow reversal in descending thoracic and abdominal aorta
 - a. Use pulsed-wave Doppler
 - b. Degree retrograde flow roughly proportional to aortic regurgitant volume
 - c. Avoid sampling flow in aortic arch branches
 - d. Requires absence of PDA, peripheral A-V fistulae, ruptured sinus of Valsalva aneurysm
 5. Volume flow at 2 intracardiac sites
 - a. Forward flow across aortic valve
 - 1) Represents total flow
 - 2) Combines forward SV and regurgitant SV
 - b. Second “reference” valve with normal flow
 - 1) Usually mitral (assumes no MR)
 - 2) Alternately pulmonic
(in adults, can be difficult to measure diameter)
 - c. Limitations
 - 1) Tedious, time-consuming
 - 2) Diameter measurements critical
 6. Proximal flow convergence method
 - a. Limited clinical validation
 - b. Difficult to image proximal jet
 - 1) Degenerative disease (fibrosis/sclerosis)
 - 2) Bicuspid valves (eccentric jets)
 7. Diastolic mitral regurgitation
 - a. More useful in acute AR
 - b. Lacks sensitivity in chronic AR
 8. Premature closure of mitral valve
 - a. More useful in acute AR
 - b. Lacks sensitivity in chronic AR

c. Not specific (1° AV block)

D. Sequential evaluation in chronic asymptomatic AR

1. Left ventricular dimensions and volume
2. LV systolic function
3. Aortic root size

E. Role of Echo-Doppler in timing of surgery

1. Echo predictors of surgical outcome

- a. LV end-systolic dimension (or volume)
- b. LV end-diastolic dimensions (or volume)
- c. LV systolic function (ejection fraction)
- d. Rate of ↑ ESD and ↓ EF over time

2. Role of exercise ejection fraction?

- a. **Not** an independent predictor (not recommended)
- b. Change in EF with exercise relate to :
 - 1) Myocardial contractility
 - 2) Severity of volume overload
 - 3) Exercise-induced changes in preload and peripheral resistance

F. Acute, severe aortic regurgitation

1. Etiologies: aortic dissection, trauma, infective endocarditis, post-balloon valvotomy or surgical commissurotomy for congenital AS
2. Echo-Doppler features (rapid equilibration of aortic and LV diastolic pressure)
 - a. Short AR diastolic half-time (<300 msec)
 - b. Short mitral deceleration time (<150 msec)
 - c. Early closure of MV
 - d. Premature closure of aortic valve

VII. Mitral Regurgitation

A. Imaging of the valve apparatus (etiology, mechanism)

1. Etiologies

- a. Rheumatic
- b. Mitral valve prolapse
- c. Ruptured chordae
- d. Infective endocarditis
- e. Ischemia, infarction
- f. Dilated cardiomyopathy
- g. Hypertrophic cardiomyopathy
- h. Calcified mitral annulus
- i. Loeffler's
- j. Connective tissue disorders
- k. Trauma
- l. Congenital
- m. Appetite-suppressants

2. Mechanisms

a. Functional classification of Carpentier

- 1) Normal leaflet motion
- 2) Excessive leaflet motion
- 3) Restricted leaflet motion

b. Specific mechanisms

- 1) Annular dilatation
- 2) Elongated and/or ruptured chordae
- 3) Abnormal shape/geometry of LV and abnormal papillary muscle orientation
- 4) Increased rigidity of leaflets

c. Jet direction as a clue to mechanism

B. Indirect signs of mitral regurgitation

1. Left ventricular dilatation
2. Left atrial dilation
3. Increased motion of aortic root on M-mode

C. Severity of mitral regurgitation

1. Flow mapping
2. CW Doppler signal intensity
3. Increased antegrade velocity due to increased transmitral volume flow
4. Systolic flow reversal in pulmonary veins
5. Volume flow at 2 intracardiac sites
6. PISA
 - a. Regurgitation flow converges toward restrictive orifice in ellipsoidal isovelocity surfaces that approximate hemispheres
 - b. Instantaneous flow at isovelocity surface = $2\pi r^2 V_a$
 - c. Effective regurgitant orifice area by PISA
 $EROA = 2\pi r^2 V_a / V_{\max-MR}$
 - d. Assumptions
 - 1) Hemispherical isovelocity surface
 - 2) Single regurgitant orifice
 - 3) Aliasing isovelocity and peak MR velocity are measured at same point in cardiac cycle
 - 4) Flow convergence not constrained by adjacent structures
 - 5) EROA is constant throughout systole

D. Chronic vs acute mitral regurgitation

E. Sequential evaluation in chronic asymptomatic MR

1. Every 6 to 12 months
2. Assess changes in LV systolic function
3. Assess LV end-systolic dimension (and/or volume)
 - a. Less load dependent than EF
 - b. LVESD <45 mm → normal post op LV function

F. Role of Echo-Doppler in timing of surgery

1. Echo predictors of surgical outcome
 - a. Ejection fraction, fractional shortening
 - b. End-systolic dimension (volume)
 - c. LV wall thickness-to-radius ratio
 - d. LV end-systolic wall stress
2. Feasibility of MV repair

3. Pulmonic hypertension
4. Role of hemodynamic stress testing (exercise or dobutamine)
 - a. Pt. with mild or moderate MR, but exercise-induced symptoms
 - b. Pt. with severe MR and minimal or no symptoms (not established role)

G. Mitral valve repair

1. Pre-op evaluation: Feasibility of repair
 - a. High likelihood of repair
 - 1) Ruptured cord to posterior leaflet (especially middle scallop)
 - 2) Small perforation
 - b. Lower likelihood of repair
 - 1) Valve calcification
 - 2) Annulus calcification
 - 3) Rheumatic involvement
 - 4) Ischemic MR
 - 5) Anterior leaflet involvement
2. Intra-op evaluation of MV repair
 - a. Successful repair
 - b. Residual MR
 - c. Failed repair
 - d. Residual leaflet prolapse
 - e. Systolic anterior motion of AML
 - f. New mitral stenosis
3. Methods of repair
 - a. Quadrangular resection
 - b. Chordal transfer, chordal reattachment
 - c. New (artificial) chordae
 - d. Sliding valvuloplasty
 - e. Annular rings
 - f. Other techniques

VIII. Mitral Valve Prolapse

A. Classification of mitral valve prolapse

1. Primary
2. Secondary
 - a. Reduced LV dimensions
(eg. ASD, HCM, pulm HTN, dehydration,
straight-back syndrome/pectus excavatum)
 - b. Rheumatic heart disease
 - c. “Flail” mitral valve leaflet(s)
 - d. CAD (pap muscle elongation)

B. Role of echo

1. Diagnosis of MVP
2. Determination of LA size, LV size/function
3. Detection/quantitation of MR
4. Risk stratification
 - a. Extent of leaflet thickness/redundancy
 - b. Degree of MR
5. Detection of associated lesions (eg. ASD)

C. M-mode echo definition

1. ≥ 2 mm posterior displacement of one or both leaflets
in mid-late systole or
2. Holosystolic posterior “hammocking” ≥ 3 mm

D. 2D-echo (no consensus on 2D echocardiography criteria)

1. Systolic displacement of one or both leaflets in PLAX view
2. Esp. if coaptation is on atrial side of annular plane
3. **Caution** if MVP observed only in apical 4-chamber view
(controversial)
4. Leaflet thickening (esp. if >5 mm)
5. Leaflet redundancy
6. Enlarged mitral annulus
7. Elongated chordae tendinae

E. Doppler

1. MR may be present or absent
2. MR often eccentric (opposite of involved leaflet)
3. MR may be late systolic

IX. Tricuspid Regurgitation

A. Imaging of the valve apparatus (etiology/mechanism)

1. Annular dilatation
2. Rheumatic valvulitis
3. Carcinoid
4. Ebstein's anomaly; other congenital
5. Endocarditis
6. Trauma
7. Radiation therapy
8. Marfan syndrome
9. TV prolapse
10. Papillary muscle dysfunction

B. Indirect signs of tricuspid regurgitation

1. Right ventricular and right atrial dilation
2. Paradoxical septal motion
3. Right ventricular volume overload

C. Severity of tricuspid regurgitation

1. Flow mapping (pulsed or color)
2. Systolic flow reversal in IVC and SVC
3. CW Doppler signal intensity

D. TR jet method to estimate pulmonary artery pressure (see Section 3, IV, D, 3)

1. May be unreliable if no "restrictive orifice" (some cases of severe TR)

X. Pulmonic Regurgitation

A. Imaging of the valve (etiology, mechanism)

1. Congenital disease
2. Endocarditis
3. Carcinoid

B. Severity of pulmonic regurgitation

1. Flow mapping
2. CW Doppler intensity
3. Shape of CW Doppler time-velocity curve
4. Holodiastolic flow reversal in main pulmonary artery

C. Clinical utility

1. Decision-making in congenital heart disease
2. Estimation of pulmonary artery diastolic pressure

XI. Prosthetic Valve

A. Types of prosthetic valves

1. Bioprosthetic valves
 - a. Stented
 - b. Stentless
2. Homograft valves
3. Mechanical valves
 - a. Ball-cage
 - b. Floating disc
 - c. Tilting disc
 - d. Bileaflet
 - e. Valve conduits

B. Fluid dynamics (normal Doppler findings)

1. Antegrade flow patterns
2. Physiologic regurgitation
3. Prosthetic valve “clicks”

C. Prosthetic valve dysfunction

1. Prosthetic valve stenosis
2. Prosthetic valve regurgitation
 - a. Central
 - b. Periprosthetic

3. Echo-Doppler clues to prosthetic valve dysfunction

- a. Increased antegrade velocity across valve
- b. Decreased valve area (continuity equation or pressure halftime)
- c. Increased intensity of CW Doppler regurgitant jet
- d. Progressive chamber dilation
- e. Recurrent or unexplained pulmonary hypertension
- f. Flow convergence on LV side of mitral

D. Other complications

1. Thrombosis, thromboembolism
2. Infection
3. Pannus (fibrous tissue ingrowth)
4. Dehiscence

E. “Routine” follow-up of prosthetic valve function

1. Value of baseline exam (appropriate)
2. Value of serial studies uncertain

F. Technical aspects and limitations

1. Acousting shadowing (“flow-masking”)
2. Reverberations
3. Overestimation of transvalvular pressure gradients
 - a. Pressure recovery phenomenon
 - b. Especially small size bileaflet mechanical values
4. Pathologic vs physiologic regurgitation
5. Normal “rocking” of sewing ring consonant with LV contractility
6. Jet length, width, area depend on multiple factors

XII. Endocarditis

A. Role of echo in diagnosis

1. Echo hallmark: vegetation
2. Echo features of vegetation
 - a. Localized echo density (mass)
 - b. Typically irregular shape
 - c. Pedunculated or sessile
 - d. Rarely impair valve motion
 - e. Often flutter or vibrate
3. Location of vegetations
 - a. Atrial side of mitral and tricuspid valves
 - b. Ventricular side of aortic and pulmonic valves
 - c. Secondary jet lesions
4. Unusual sites of vegetations
 - a. Chordae tendinae
 - b. Mural endocardium
 - c. Eustachian valve
 - d. Pacemaker wires
 - e. Calcified mitral annulus
 - f. Mural thrombus
5. Diagnostic accuracy of echo
 - a. M-mode
 - b. 2D-echo (TTE)
 - c. Transesophageal
6. Mimics of vegetations
 1. Myxomatous degeneration
 2. Ruptured or redundant chordae
 3. Focal, nonspecific thickening or calcium deposits
 4. Retained mitral leaflets/apparatus post MV replacement
 5. Lambl's excrescences and valve "strands"
 6. Sutures, strands on prosthetic sewing rings
 7. Tumors, thrombi

B. Hemodynamic sequele

C. Prognosis

1. CHF, mortality, need for surgery, embolism
2. Size of vegetation and risk of embolism

D. Complications

1. Paravalvular abscess
2. Intracardiac fistulae
3. Mitral valve aneurysm
4. Aneurysms of mitral aortic intervalvular-fibrosa region
5. Dehiscence of prosthetic valve
6. Obstruction due to bulky vegetation (rare)
7. Purulent pericarditis

E. Special considerations

1. Natural history of vegetations
2. Active vs healed vegetations
3. Nonbacterial thrombotic endocarditis
4. Infections of pacemaker and catheters
5. Surgery for endocarditis: role of echo
 - a. Indications
 - b. Timing of surgery
 - c. Intra-operative echo

F. Indications for TEE

XIII. Valvular Heart Disease Associated with Systemic Conditions

A. Connective tissue diseases

1. Systemic lupus erythematosus (SLE)
 - a. General comments
 - 1) Reported prevalence varies (10-100%)
 - 2) Anatomic and functional abnormality usually mild
 - 3) Often clinically silent
 - 4) Valve disease does not correlate with clinical features of SLE

b. Echo findings

- 1) Leaflet thickening (fibrosis)
- 2) Valve masses (Libman-Sacks vegetations)
 - a. Almost exclusively mitral and aortic
 - b. Usually small ($<1 \text{ cm}^2$)
 - c. Usually no independent motion
 - d. Usually irregular borders
- 3) Valve regurgitation
- 4) Valve stenosis (rare)

2. Ankylosing spondylitis

- a. Nonspecific thickening of aortic and mitral leaflets
- b. Thickening of base of anterior mitral leaflet (“subaortic bump”)
- c. Increased echogenicity of posterior aortic wall
- d. Aortic root dilatation

Section 8: Diseases of the Myocardium (nonischemic)

I. Cardiomyopathies

A. Dilated cardiomyopathy

1. Echo features
2. Associated findings
 - a. Mitral regurgitation
 - b. Thrombi (see Section 12, II)
 - c. Other chamber enlargement
3. Prognostic role of echo
 - a. Ejection fraction
 - b. Deceleration time
 - c. RV function

B. Hypertrophic cardiomyopathy

1. Epidemiology
 - a. Prevalence, incidence
 - b. Associated risk factors for sudden death
 - c. Presentation
 - d. Genetics
2. Anatomic features
 - a. Varied patterns of myocardial hypertrophy (pleomorphic)
 - 1) Asymmetric septal hypertrophy (“classic”)
 - 2) Basal septal hypertrophy (DUST – disproportionate upper septal thickening)
 - 3) Mid-ventricular (with/without apical aneurysm)
 - 4) Apical
 - 5) Concentric
 - 6) Others

- b. Non-dilated LV cavity
- c. Narrowed LVOT diameter
- d. Finely granular speckled appearance of myocardium
- e. Mitral apparatus
 - 1) Anterior displacement of mitral apparatus
 - 2) Elongated anterior mitral leaflet (AML)
 - 3) Increased area of AML
 - 4) Calcified mitral annulus
- f. Mid-systolic closure of aortic valve
- g. Upper septal endocardial thickening (“contact lesion”)
- h. Atrial dilatation (especially LA)

3. Physiologic features

- a. LV outflow tract gradient
- b. Mitral regurgitation
- c. Midcavitary vs LVOT obstruction
- d. Diastolic dysfunction

4. LVOT obstruction (dynamic)

- a. May, or may not be present
- b. Late-peaking
- c. Reliably assessed by Bernoulli equation
- d. Narrowed LVOT and SAM-septal contact
- e. Increased with certain maneuvers
 - 1) Standing
 - 2) Valsalva
 - 3) Amyl nitrate

5. Spectrum of dynamic obstruction

- a. Classic mitral-septal contact with septal hypertrophy
- b. Mid-cavity muscular obstruction
 - 1) Aliasing begins more apically
 - 2) Steep late-systolic rise in velocity
- c. Late-systolic cavity obliteration
 - 1) Concentric LVH with hyperdynamic contractility
 - 2) May be induced by dobutamine

- d. Recently described mid-cavitary obstruction by hypertrophic papillary muscles inserting directly on the MV
6. Systolic anterior motion of mitral valve (SAM)
- a. Anterior motion of distal leaflet tip
 - b. One or both leaflets
 - c. Degree of SAM-septal contact corresponds to gradient
 - d. Mechanism(s)
 - 1) Venturi effect
(high outflow velocities in narrowed tract)
 - 2) Anterior pap muscle displacement and leaflet elongation
 - e. Other situations
 - 1) Mitral valve repair
 - 2) Aortic valve replacement (aortic stenosis)
 - 3) Hypovolemia
 - 4) Endogenous and exogenous catecholamines
7. Mitral regurgitation
- a. Virtually always seen with obstructive SAM
(disrupted coaptation)
 - b. Otherwise, milder and less frequent
 - c. Eccentric (posterolateral), late systolic
 - d. Occasionally independent of HOCM (primary MR)
8. Diastolic dysfunction (complex determinants)
- a. Characterized by:
 - 1) Impaired relaxation
 - 2) Decreased compliance
 - 3) Increased LVEDP
 - b. Often decreased mitral E/A ratio (abnormal LV relaxation)
 - c. Normal filling pattern also common (does not imply absence of intrinsic LV diastolic abnormalities)
 - 1) Normalization by MR
 - 2) Normalization by increased LA pressure

- d. Poor correlation between decel time and LA pressure
- e. Presumed cause of symptoms in pts with no obstruction
- f. Clinical significance in asymptomatic pts unknown

9. Limitations and pitfalls of Echo

- a. HCM can be mimicked
 - 1) Chronic HTN (esp pts with renal failure)
 - 2) Cardiac amyloid
 - 3) Pheochromocytoma
 - 4) Friedrich's ataxia
 - 5) Inferior MI with previous LVH
- b. Dynamic LVOT obstruction not specific
 - 1) Hyperdynamic LV systolic function (especially if basal septal hypertrophy)
 - a) Anemia, fever
 - b) Volume depletion
 - c) Catecholamines
 - 2) Hypertensive "HCM" of the elderly
 - 3) Post-op period (volume depletion and inotropic agents)
 - 4) Aortic stenosis after valve replacement
 - 5) Acute antero-apical MI, esp. if pre-existing basal septal hypertrophy
- c. Apical HCM sometimes missed
- d. False-positive diagnosis due to RV papillary muscle, moderator band, and/or prominent RV trabeculations overlying ventricular septum

10. Role of echo in evaluation of therapy

11. Role of echo for DDD pacing

- a. Optimization of AV interval
 - 1) Too short: Increased LA pressure
 - a) Increase of mitral E
 - b) Early cessation of mitral E

- 2) Too long: Inadequate relief of LVOT gradient
(also fusion of QRS complex)
- b. Placement of pacemaker lead
 - 1) Fluoro may be inadequate
 - 2) Critical position
 - a) Optimal is RV apex
 - b) Deterioration of RV septum

12. Role of echo for alcohol septal ablation

- a. Patient selection
 - 1) LVOT gradient
 - 2) Confirmation, localization septal hypertrophy
 - 3) Location of SAM septal contact
- b. Guidance of procedure
 - 1) Infuse echo contrast in septal perforator(s)
 - 2) Confirm appropriate distribution of septal perforator
 - 3) Exclude perfusion of RV, pap muscle(s), other areas of LV
 - 4) Hemodynamic results
- c. Follow-up
 - 1) LVOT gradient
 - 2) Regression of hypertrophy
 - 3) MR
 - 4) Diastolic filling

13. Role of Intra-op echo for surgical myotomy-myectomy

- a. Determine site and extent of resection
 - 1) Accurate measurement septal thickness
 - 2) Distance from aortic annulus
 - 3) Point of SAM-septal contact
- b. Assess immediate results
 - 1) Extent of resection (thinning of septum)
 - 2) Widening of LVOT
 - 3) Resolution of SAM

- 4) Elimination/reduction of gradient
 - a) Not always feasible with TEE
 - b) TEE: deep transgastric views
 - c) TEE: 120° - 130° long-axis view

- 5) Elimination/reduction of MR

c. Detect/exclude complications

- 1) Septal perforation
- 2) Worsening of MR
- 3) If muscle bridge unroofed:
 - a) Sparing and occlusion of LAD
 - b) Inadvertent RV perforation

C. Restrictive cardiomyopathy (RCM)

1. Etiologies

a. Primary

- 1) Idiopathic
- 2) Loeffler's eosinophilic endomyocardial disease
- 3) Endomyocardial fibrosis

b. Secondary

- 1) Amyloid heart disease
- 2) Hemochromatosis
- 3) Heart muscle disease occasionally presenting with restrictive features
 - a) Post irradiation heart disease
 - b) Carcinoid heart disease
 - c) Doxorubicin/daunorubicin toxicity
 - d) Progressive systemic sclerosis

2. Typical 2D-echo findings

- a. Small to normal LV cavity size
- b. Often thick LV walls
- c. Normal, near-normal LV function
- d. Dilated atria

3. Doppler: restrictive physiology (see Section 5, II, D, 4 and 5)
4. Differentiating RCM vs constrictive pericarditis (see Section 9, III, C)

D. Infiltrative cardiomyopathy (overlaps with RCA)

1. Classification

a. Interstitial

- 1) Amyloid
- 2) Hemochromatosis
- 3) Sarcoid
- 4) Malignancy

b. Storage

- 1) Glycogen storage
- 2) Lipid storage

2. Echo features (using with specific disease)

E. Arrhythmogenic Right Ventricular Dysplasia (ARVD)

1. Definition

- a. Primary cardiomyopathy of unknown cause
- b. Characterized by progressive loss of RV myocardium with replacement by peculiar fatty or fibro-fatty tissue
- c. Associated with ventricular arrhythmias and sudden death in young

2. 2D-echo findings

- a. Dilated RV
- b. Aneurysms, outpouchings of RV
(distributed in RV inflow, apex, infundibulum)
- c. Focal RV wall thinning
- d. Abnormal global, regional RV systolic wall motion
- e. Abnormal tissue composition – RV muscle replaced by fat
(better detected MRI)
- f. Lesser involvement of LV (until late)

3. Other arrhythmogenic RV cardiomyopathies
 - a. RV outflow tract tachycardia
 - b. Benign extrasystoles
 - c. Uhl's anomaly
 - d. Biventricular dysplasia

- F. Myocardial disease associated with neuromuscular disorders

- G. Myocardial disease due to toxic agents and infectious diseases

- H. Cardiac abnormalities resulting from trauma

- I. Effect of systemic illnesses on the heart
 1. Anemia
 2. Connective tissue disorders
 3. Thyroid disorders
 4. Hemachromatosis
 5. Others

- J. Cardiac transplantation
 1. Allograft morphology and function
 2. Complications
 - a. Pericardial effusion
 - b. Rejection
 - c. Coronary artery disease
 - d. RV systolic dysfunction

 3. Echo-Doppler detection of rejection
 - a. Deceleration time, IVRT
 - b. Tissue Doppler

Section 9. Systemic and Pulmonary Hypertensive Heart Disease

I. Systemic Hypertension

A. Physiology/hemodynamics

1. Increased afterload leads to ventricular hypertrophy and increased mass
2. Increased hypertrophy/mass leads to diastolic dysfunction

C. Echocardiographic findings

1. Increased LV mass and mass index
2. Hypertrophy
3. Enlarged left atrium (due to increased LVEDP)
4. Dilated aortic root
5. Mitral annular calcification
6. Right ventricular hypertrophy

D. Diastolic abnormalities

1. Abnormal relaxation
 - a. Early
 - b. Minimal or no symptoms at rest
2. Pseudonormalization
 - a. Later stage
 - b. Minimal or no symptoms at rest
 - c. Symptoms with mild/moderate exertion
3. “Restrictive” pattern (High LA pressure pattern)
 - a. Substantial increase in LA pressure
 - b. Symptoms at rest or with minimal exertion
 - c. Poor prognosis

E. Prognostic value of echo

F. Efficacy of medical therapy (regression of hypertrophy)

G. Complications of hypertension

- a. Aortic insufficiency
- b. Aortic dissection

H. Rule out secondary hypertension (coarctation)

I. Hypertensive hypertrophic cardiomyopathy of the elderly

II. Pulmonary Heart Disease (Cor pulmonale)

A. Physiology/hemodynamics

B. Role of echocardiography

1. Detection of pulmonary hypertension
2. Detection of occult pulmonary hypertension (exercise echo)
3. Quantitation of pulmonary hypertension
4. Determine etiology of pulmonary hypertension
5. Evaluate end-effects of pulmonary hypertension
6. Determine prognosis

C. Echo-findings

1. RV dilatation, hypertrophy
2. RV function
3. Abnormal septal geometry
4. Dilated pulmonary artery
5. Pulmonic valve motion

D. Assessment of pulmonary hypertension (see Section 3, IV, D)

E. Chronic vs acute cor pulmonale

F. Acute pulmonary embolism

1. Acute RV pressure overload
2. RV dilatation and dysfunction
3. Thrombus in right heart (residual or “in-transit”)

Section 10. Pericardial Disease

I. Normal Pericardial Anatomy

II. Pericardial Effusion

- A. Detection of pericardial effusion
- B. Differentiation between pericardial and pleural effusion
- C. Differentiation between pericardial effusion and epicardial fat
- D. Quantitation of pericardial fluid
- E. Echo-Doppler diagnosis of cardiac tamponade
 - 1. Right atrial systolic collapse
 - 2. Right ventricular diastolic collapse
 - 3. Reciprocal changes in ventricular volumes
 - 4. Respiration variation in RV and LV diastolic filling
 - 5. Plethora of inferior vena cava
- F. Echo-guided pericardiocentesis (see Section 18, I, B)

III. Constrictive Pericarditis

- A. Pathophysiology
 - 1. Elevated diastolic pressures
 - 2. Rapid early diastolic filling that stops abruptly as limits of ventricular expansion achieved (Dip and plateau in diastolic pressure tracing)
 - 3. Respiratory changes in pleural pressures not transmitted to heart (dissociation b/w intrathoracic and intracardiac pressures)
 - 4. Increased ventricular interaction
- B. Echo-Doppler diagnosis of pericardial constriction
 - 1. M-mode and 2D-echo
 - a. Pericardial thickening
 - b. Normal LV size and contractility
 - c. Atria normal or enlarged
 - d. “Flattened” motion of posterior wall in diastole

- e. Abrupt posterior motion of ventricular septum in early diastole (septal “bounce”)
- f. Dilated IVC and hepatic veins
- g. Premature diastolic opening of pulmonic valve

2. Doppler

- a. Prominent y descent on hepatic vein or SVC flow pattern
- b. LV inflow shows prominent E-wave with rapid early diastolic deceleration slope
- c. Increase in LV-IVRT by >20% on first beat after inspiration
- d. Respiratory variations in RV/LV diastolic filling (>25%) with increase in RV filling and decrease in LV filling with inspiration
- e. Pulmonary venous flow: systolic \geq diastolic flow with resp. variation
- f. Hepatic vein: decrease or loss of diastolic filling with marked expiratory reversal

C. Differentiating constriction vs restrictive cardiomyopathy

1. Restrictive cardiomyopathy (RCM)

- a. Lack of respiratory variation (MV/TV/IVRT)
- b. Shorter deceleration time
- c. Diastolic mitral regurgitation
- d. Reduced mitral and tricuspid A waves during apnea
- e. Tissue Doppler imaging
 - 1) RCM relaxation abnormal
 - 2) CP relaxation normal
- f. Color M-mode
 - 1) RCM prolonged slope
 - 2) CP rapid slope
- g. Concordance of LV and RV pressures with respiration

2. Constrictive pericarditis (CP)

- a. Thick pericardium
- b. Inspiratory decrease in mitral inflow
- c. Inspiratory increase in tricuspid inflow
- d. Rapid color M-mode slope (> 100 cm/sec)
- e. Increased TDI E-wave annular velocity (> 12 cm/sec)
- f. Discordance of LV and RV pressures with respiration

3. Pitfalls in differentiating RCM vs CP

- a. COPD (false positive)
- b. Sample volume placement
- c. Depth of respiration (false positive with increased respiratory effort)
- d. Atrial fibrillation, other irregular rhythms
- e. False negative
 - 1) High filling pressure
 - 2) Localized constriction

IV. Pericardial Cysts

V. Congenital Absence of Pericardium

I. Basic Embryology

A. Primitive cardiac formation

1. Formation from primitive vascular tube
2. Sinus venosus
3. Cardiac loop
4. Aortic arches
5. Septation
6. Valve formation

B. Comparison of fetal and postnatal circulation

II. Segmental Approach

A. Cardiac location

1. Position (in chest)
2. Orientation (position of cardiac apex)
 - a. Levocardia
 - b. Dextrocardia
 - c. Mesocardia

B. Visceral situs

1. Solitus
2. Inversus
3. Ambiguous

C. Atrial situs (RA right or left-sided; LA right or left-sided)

1. RA characteristics
 - a. IVC and SVC drain into RA
 - b. Eustachian valve often seen
 - c. Wide spade-like RA-appendage

2. LA characteristics

- a. Pulmonary veins drain into LA
- b. Overlap of septum primum onto the superior atrial septum occurs on LA side
- c. Long finger-like LA-appendage with narrow neck

D. Determine ventricular morphology

1. RV morphology

- a. Triangular shape
- b. Coarse trabeculations
- c. Three papillary muscles
- d. Moderator band
- e. Tricuspid valve attachment

2. LV morphology

- a. Elliptical shape
- b. Smooth, fine trabeculations
- c. Two papillary muscles
- d. Mitral valve attachment

3. A-V valve identification

- a. Mitral valve
 - 1) Two leaflets
 - 2) Two papillary muscles
 - 3) Inserts more superiorly on septum
- b. Tricuspid valve
 - 1) Three leaflets
 - 2) Three papillary muscles
 - 3) Inserts more apically on septum

E. Delineate atrioventricular connections

- 1. AV concordance
- 2. AV discordance
- 3. Double inlet
- 4. Straddling/override
- 5. Common inlet

F. Identify great arteries and ventriculoarterial connection

1. VA concordance
2. VA discordance
3. Double outlet
4. Great artery position (relative)

III. Hemodynamics (see Section 3)

- A. Pressure gradients
- B. Valve areas
- C. Pulmonary artery pressure
- D. Quantitation of flow
- E. Quantitation of shunts

IV. Outflow Obstruction

A. Valvular aortic stenosis (See also Section 6, I)

1. Unicuspid valve
2. Bicuspid valve (1-2% of population; male predominance (4:1)
 - a. Stenosis, regurgitation
 - b. Endocarditis
 - c. Coarctation
 - d. Dilated ascending aorta, dissection
3. Tricuspid valve

B. Subvalve aortic stenosis

1. Anatomic variables
 - a. Discrete membrane
 - b. Fibro-muscular ridge
2. Aortic regurgitation
3. Associated conditions
 - a. Supramitral ring
 - b. Coarctation

4. Regrowth after resection

C. Supravalve aortic stenosis

1. Seldom occurs in isolation
2. Rarely localized
3. Usually diffuse, involving the major arteries to varying degree
4. Aortic regurgitation common
5. Associated with Williams syndrome

D. Coarctation of the aorta

1. Suprasternal view for anatomy and presence of turbulent flow
2. Doppler: measure Vmax and diastolic flow pattern
3. Measure LV systolic and diastolic dimensions and LV function
4. Quantify LVH
5. Associated with bicuspid valve and ascending aortic aneurysm
6. Post-repair
 - a. Recoarctation
 - b. Aneurysm of repair site (MRI may be better)

E. Right ventricular outflow tract obstruction

1. Levels of obstruction
 - a. Valvular
 - 1) Most valvular PS isolated
 - 2) In 15% valve is dysplastic as well as stenotic
 - 3) In adults, valve may calcify after 4th decade
 - 4) Echo features
 - a) Valve leaflet thin or thick
 - b) May be bicuspid or unicuspid
 - c) May “dome” in systole
 - d) Post-stenotic dilation of main PA
 - e) Pressure hypertrophy of RV
 - f) May be secondary infundibular stenosis
 - b. Subvalve (infundibular)
 1. Usually in combination with other lesions
 - a. VSD
 - b. Tetralogy of Fallot
 2. May occur in association with subaortic stenosis

c. Supravalve

1. Seldom occurs in isolation from valvular abnormality
2. Associations

- a. Tetralogy of Fallot
- b. Williams syndrome
- c. Noonan syndrome
- d. VSD
- e. Arteriohepatic dysplasia

d. Peripheral (branch) stenosis

2. Echo Doppler evaluation

a. Document level(s) of obstruction

- 1) In general, echo poor for PA anatomy, especially distal lesions
- 2) Best views

- a) Parasternal short axis of great arteries
- b) Low parasternal 4-chamber view with upward angulation

- 3) Color Doppler can be helpful
- 4) TEE can be helpful

- b. Quantify severity of obstruction
- c. Identify associated abnormalities

3. Post-intervention (balloon valvuloplasty or surgery)

- a. Residual or recurrent obstruction
- b. Pulmonic regurgitation
- c. Deterioration of RV function

V. Abnormal Intracardiac Communications

A. Atrial septal defect

1. Types

a. Secundum

- 1) Most common type
- 2) At fossa ovalis

b. Primum (partial AV canal; endocardial cushion defect)

- 1) Lies antero-inferior to fossa ovalis
- 2) Often assoc'd with cleft mitral valve

c. Sinus venosus

- 1) Lies posterior to fossa ovalis
- 2) Usually assoc'd with anomalous connection of right pulmonary vein(s) to RA

d. Coronary sinus

- 1) Involve anticipated site of coronary sinus ostium
- 2) The coronary sinus, if present, straddles the defect

2. Pathophysiology, hemodynamics

- a. L-R shunt because compliance $RV < LV$
(not because LA pressure slightly higher than RA pressure)
- b. Pulmonary blood flow may be 2-4 times normal
- c. RV volume overload
- d. Sometimes RV pressure overload
- e. Any condition causing reduced LV compliance (eg. LVH, MI, cardiomyopathy) tends to increase L-R shunt through ASD
- f. MS and/or MR increases L-R shunt through ASD

3. 2D-echo findings

- a. Type and size of defect RV size, function, volume overload
- b. Pulmonary arteries became dilated
- c. Eventually tricuspid and pulmonary annuli also dilate

- 4. Doppler findings
 - a. Spectral
 - 1) Flow begins in early systole, continues almost through cardiac cycle with a broad peak in late systole and early diastole
 - 2) PA pressure (from tricuspid regurgitant signal)
 - 3) Qp/Qs
 - b. Color
- 5. Contrast
- 6. TEE: If not provided by TTE
 - a. Detect size and size of rims
(to assess suitability for device closure)
 - b. Assess pulmonary venous return
 - c. Evaluate valves
- 7. Post ASD repair
 - a. Residual shunt
 - b. Residual pulmonary hypertension
- 8. Associated lesions
 - a. Secundum: mitral valve prolapse
 - b. Primum
 - 1. Cleft anterior mitral leaflets
 - 2. Inlet VSD
 - 3. Partial attachment of MV to IVS
 - c. Sinus venosus: partial anomalous pulmonary venous drainage

B. Ventricular septal defect

- 1. Types
 - a. Perimembranous (subaortic)
 - 1) Most common type of VSD
 - 2) Involves or is adjacent to membranous septum

- b. Subpulmonic (infundibular; supracristal)
 - 1) Involves RV outflow tract (conus or infundibulum)
 - 2) Frequently partially occluded by a cusp of the aortic valve
 - 3) May cause aortic insufficiency over time
- c. Muscular
 - 1) Single or multiple
 - 2) Involves inflow or trabeculated (apical) ventricular septum
- d. Atrioventricular canal (inlet VSD)
 - 1) Involves inflow portion of septum
 - 2) Always assoc'd with AV valve abnormalities

2. Physiology/hemodynamics

- a. L-R shunt occurs because $PVR < SVR$
(not because LV pressure higher than RV pressure)
- b. Hemodynamic severity
 - 1) Small
 - a) Pulm/systemic pressure ratio <0.3
 - b) $Qp/Qs <1.4$
 - c) Diameter $<25\%$ of aortic anulus
 - 2) Moderate
 - a) Pulm/systemic pressure ratio <0.3
 - b) Qp/Qs 1.4 to 2.2
 - c) Diameter 25-50% of aortic anulus
 - 3) Large
 - a) Pulm/systemic pressure ratio >0.3
 - b) $Qp/Qs >2.2$
 - c) Diameter $>50\%$ of aortic anulus
 - 4) Eisenmenger
 - a) Pulm/systemic pressure ratio >0.9
 - b) $Qp/Qs <1.5$

3. 2D-echo

- a. Type and size of defect
- b. Ventricular size and function (increased with large shunt)
- c. Left atrial size (increased with large shunt)

4. Doppler

a. Spectral

- 1) Interventricular gradient
- 2) QP/QS
- 3) Pulmonary artery pressure

b. Color

- 1) Shunt flow
- 2) Other abnormalities

5. Contrast echo

6. Associated lesions

- a. Approx. 25-50% are isolated defects
- b. PDA, ASD, coarctation
- c. Integral part of certain anomalies
(eg. tetralogy, truncus, some transpositions)
- d. If subaortic or subpulmonic VSD, check for prolapse
of right coronary cusp and aortic regurgitation

7. Post VSD repair

- a. Patch
- b. Residual shunt
- c. Residual pulmonary hypertension

C. Patent ductus arteriosus

1. Physiology/hemodynamics

- a. Small PDA: Negligible hemodynamic change
 Normal size LV
 No pulmonary hypertension
- b. Moderate PDA: LV enlargement
 Some pulmonary hypertension (usually reversible)

- c. Large PDA: Usually leads to Eisenmenger physiology
Enlargement of LV and LA
Rare in adults
Aneurysm of duct is uncommon but important complication

2. 2D-echo findings

- a. Left ventricular volume overload
- b. Dilated left atrium

3. Doppler

- a. Detection by color/spectral Doppler
 - 1) Short-axis of great arteries and distal main PA
 - 2) Aortic arch view (measure size of PDA : length and width)
 - 3) Ductal view (high parasternal sagittal plane)
- b. PA pressure (functional significance)
- c. Spectral recording of ductal flow signal to assess PA pressure (Normally continuous flow with systolic peak)
- d. Qp/Qs

4. Associated lesions (PDA is usually an isolated lesion)

- a. Endocarditis
- b. Seldom other (eg. VSD)

5. Post PDA repair

- a. Residual shunt

D. Atrioventricular septal defects

1. Types

- a. Partial (no VSD)
- b. Complete (Inlet VSD)

2. Associated lesions

3. Post-op
 - a. Residual ASD
 - b. Residual VSD
 - c. LV to RA shunt
 - d. AV valve insufficiency
- E. Partial anomalous pulmonary venous drainage
 1. One or 2 pulmonary veins connect to RA
 2. 80% of pts with sinus venosus ASD; 10% of pts with secundum ASD
 3. Common connections
 - a. RUPV to RA or SVC (>90% cases)
 - b. Left PV connecting to innominate vein
 - c. Right PV connecting to IVC

VI. Persistent Left Superior Vena Cava

- A. Commonly drains into RA by way of coronary sinus; may drain directly to left atrium (uncommon)
- B. Echo findings
 1. Markedly dilated coronary sinus
 2. Suprasternal view shows left SVC
 3. Left arm contrast injection shows up in coronary sinus first, then RA

VII. Other Anomalies of Systemic Venous Return

- A. Interrupted IVC → SVC, LSVC
- B. Right or left SVC → LA

VIII. Ebstein's Anomaly

- A. Wide spectrum of anatomic and functional abnormalities of the morphologic tricuspid valve
- B. Echo features
 1. Apical displacement of septal and posterolateral leaflets of TV into the RV
 2. Resultant "atrialization" of RV inflow to varying degrees
 3. Tethering and mobility of anterior leaflet important for repair
 4. Enlargement of RA
 5. Varying impairment of RV function
 6. Varying impairment of LV function

C. Doppler features

1. Tricuspid regurgitation
 - a. Varying degrees
 - b. Inferiorly displaced origin of TR jet
2. Shunt at atrial level (approx. 50%); Either secundum ASD or pfo
3. Varying degrees of anatomic or physiologic RV inflow or outflow tract obstruction

D. Associated lesions

1. ASD
2. Pulmonic stenosis
3. Less common:
 - a. Coarctation
 - b. MV prolapse

E. Echo-Doppler: Selection of patients for surgery

1. Determine anatomic severity
 - a. Degree right-sided enlargement
 - b. Degree RV dysfunction
 - c. Degree TR
2. Potential of TV for surgical repair
 - a. Mobility of anterior leaflet
 - b. Insertion of anterior leaflet into ventricular outlet
3. Presence of atrial communication

F. Indication for TEE – if anatomic information not provided by TTE

G. Post-repair evaluation

1. Recurrent TR
2. Degeneration of a bioprosthetic valve
3. RV function (may worsen)

IX. Tetralogy of Fallot

A. Pathophysiology (varies depending upon degree of RVOT obstruction)

1. Mild RVOTO: increased pulm blood flow (acyanotic Tet)
2. Significant RVOTO: consequent R-L shunt across VSD

B. Echo features

1. Large malalignment type subaortic **VSD**
(due to anterocephalad deviation of outlet septum)
2. **RVOT obstruction** (infundibular, valvular, or both)
 - a. Some pts have supravalve PS
 - b. Some pts have branch (peripheral) PS
3. Large “**overriding**” **aorta** (deviated to right)
4. **RVH** (secondary to RVOT obstruction)

C. Associated Features

1. Anomalous coronary arteries (esp. LAD from right coronary – 5%)
2. ASD (15%)
3. Additional muscular VSDs (2%)
4. Right aortic arch (25%)

D. Surgical correction

1. Patch closure of VSD
2. Removal of obstructive RV outflow tract muscle
3. Removal of dysplastic or hypoplastic pulmonic valve (if needed)
4. Augmentation of RVOT and/or PV annulus

E. Post-surgical evaluation

1. Residual RVOT obstruction
2. Pulmonic regurgitation
3. Assessment of RV size, function
 - a. RVVO
 - b. Dilated TV annulus, TR
4. Diameter of MPA, LPA, and RPA
5. RVOT aneurysm
6. Residual VSD
 - a. Patch leak
 - b. VSD peak velocity
7. Aortic root size and AI
8. LV size and function

X. Complex Congenital Lesions

A. Conotruncal abnormalities

1. Transposition (“ventriculo-arterial discordance”)

a. D-transposition of great arteries

1) Two forms

- a) With intact ventricular septum
- b) With VSD (usually membranous)

2) Associated defects

- a) ASD
- b) VSD (usually perimembranous)
- c) Pulmonic stenosis
- d) PDA
- e) Coarct of aorta

3) Echo features

- a) Aorta arises from RV
- b) Aorta located anterior and to right of PA
- c) PA arises from LV
- d) Aorta and PA course parallel to each other

b. L-transposition (corrected transposition)

1) Associated defects

- a) ASD
- b) VSD
- c) Pulmonic stenosis
- d) Dysplastic left (tricuspid) AV valve
- e) Straddling left AV valve
- f) Superio-inferior ventricles
- g) Aortic/subaortic stenosis
- h) Coarctation/arch anomalies
- i) Visceral situs abnormalities (situs inversus)
- j) Rhythm abnormalities

2) Echo features

- a) LA committed to a trabeculated RV
- b) Aorta arises from RV
- c) Aorta located anterior and to left of PA
- d) PA arises from LV
- e) TV is within body of RV; MV within LV

2. Double outlet right ventricle
3. Truncus arteriosus

B. Single ventricle

1. Tricuspid atresia
2. Double inlet ventricle
3. Hypoplastic left heart syndrome
4. Pulmonic atresia/intact septum (HRHS)

C. Other

1. Total anomalous pulmonary venous return
 - a. Supracardiac
 - b. Infradiaphragmatic
 - c. Cardiac/coronary sinus
2. Atrial membranes
 - a. Cor triatriatum
 - 1) Linear echo in LA in long-axis and 4-chamber views
 - 2) Show membrane orifice and increased velocity with color Doppler
 - 3) Measure Vmax and mean gradient with spectral Doppler
 - 4) Assess RV pressure from TR jet
 - b. Supravalve mitral membrane
 - c. Cor triatriatum dexter
3. Aorto-pulmonary window

XI. Anomalous Origin or Course of Coronary Arteries

- A. Origin of LM or LAD from pulmonary trunk
 - 1. May be segmental LV dysfunction
 - 2. May be pap muscle dysfunction and MR
 - 3. May present as a LV aneurysm

- B. Ectopic origin of either RCA or LCA from opposite sinus of Valsalva
 - 1. Ectopic artery passes between aorta and RVOT
 - a. May be hypoplastic or kinked
 - b. May be mechanically compressed by great vessels (esp during exercise or HTN)

 - 2. May present in children and young adults (<40 years)
 - a. Exertional chest pain
 - b. Arrhythmias
 - c. Sudden death

- C. Single coronary artery ostium
- D. Separate ostia for LAD and LCx
- E. Hypoplastic coronary arteries
- F. Tunnel coronary arteries

XII. Congenital Abnormalities of the Aorta

- A. Left aortic arch (normal)
- B. Right aortic arch
 - 1. Incidence in congenital heart disease
 - 2. Types

- C. Double aortic arch
- D. Circumflex aorta
- E. Cervical aortic arch
- F. Coarctation of aorta (see Section 10, IV, D, pg 40)

XII. Operations for Congenital Heart Disease (Seen in Surviving Adults)

A. Shunts

1. Blalock – Taussig

- a. Anastomosis of subclavian artery to PA (or conduit to PA and subclavian remains intact)
- b. Used to increase pulmonary blood flow
- c. Echo-Doppler
 - 1) Imaged from suprasternal or high parasternal window
 - 2) Spectral Doppler to assess gradients
 - 3) Desc'g aortic flow velocity profile helpful in assessing shunt (ratio of diastolic flow reversal to forward flow)

2. Blalock-Hanlon

- a. Surgical atrial septostomy or removal of atrial septum
- b. Largely replaced by blade-and-balloon septostomy
- c. Creates atrial mixing
 - 1) TGA (early palliation)
 - 2) Mitral atresia
 - 3) Complex congenital heart disease

3. Brock procedure

- a. Closed pulmonary valvotomy
- b. Precedes days of balloon valvuloplasty

4. Rashkind balloon

- a. Percutaneous atrial septostomy
- b. Increases mixing of blood
 - 1) TGA
 - 2) Tricuspid atresia

5. Potts

- a. Descending aorta to left PA
- b. To increase pulmonary flow (alternate to Blalock)
 - 1) TGA
 - 2) Tricuspid atresia

6. Waterston

- a. Ascending aorta to right PA
- b. To increase pulmonary flow (alternate to Blalock)
- c. Rarely done anymore

7. Glenn

- a. SVC to divided right PA
- b. Increases pulmonary flow
- c. Classic Glenn rarely done; replaced by bi-directional Glenn
 - 1) Tricuspid atresia
 - 2) Pulmonic atresia

B. Closures

1. ASD

- a. Primary or patch closure
- b. Device closure

2. VSD – primary or patch closure

3. PDA

- a. Ligation \pm division of PDA
- b. Coil or device closure

4. Endocardial cushion defect repair

- a. Closure of ASD and VSD
- b. Repair of AV valve abnormalities

C. PA banding

1. Decreases PA flow and pressure
 - a. May be external (closed heart)
 - b. May be intraluminal disc (open heart)
2. For large left-to-right shunts
3. Echo-Doppler
 - a. Linear structure transversely across main PA
 - b. Doppler to assess gradients
 - c. Residual narrowing (due to scarring) may be seen after band removal

D. Atrial baffles

1. Mustard
 - a. Atrial switch with intra-atrial baffle made of pericardium or Dacron
 - b. Reestablishes proper flow sequence to PA and aorta in TGA
 - c. Replaced by arterial switch at most centers
2. Senning
 - a. Atrial switch using RA free wall and atrial septal tissue
 - b. Reestablishes proper flow sequence to PA and aorta in TGA
 - c. Replaced by arterial switch at most centers

E. Relief of stenosis

1. Coarctation
 - a. Subclavian flap angioplasty
 - b. End-to-end anastomosis
 - c. Patch enlargement
 - d. Gore-Tex graft
 - e. Balloon dilatation
2. Pulmonic valvotomy
 - a. Brock trans-RV approach (closed pulmonary valvotomy and infundibulotomy); Rarely done anymore
 - b. Direct surgical repair
 - c. Balloon dilatation

3. Aortic valvotomy
 - a. Direct surgical valvotomy
 - b. Balloon dilatation
 - c. CUSA (cavitating ultrasonic aortic valvuloplasty)
4. Mitral valvotomy
 - a. Open commissurotomy
 - b. Balloon dilatation
5. Konno procedure
 - a. Aortic annular enlargement with aortic valve replacement
 - b. Used for LVOT obstruction not amenable to valvotomy
6. Ross procedure
 - a. Pulmonary autograft to aortic position
 - b. Pulmonary homograft to pulmonary position

F. Great vessel switch (Jatene procedure)

1. Switch of aortic root and PA trunk
2. Coronaries transposed to neo-aorta

G. Conduits

1. Fontan procedure
 - a. Several variations
 - 1) Anastomosis or conduit between RA and PA
 - 2) Anastomosis of SVC to RPA in bi-directional fashion with intraatrial tunnel
 - b. Normalizes pulmonary flow
 - 1) TGA
 - 2) Univentricular heart
 - 3) Double-inlet ventricle with pulmonic stenosis
 - 4) Tricuspid atresia

c. Echo-Doppler

- 1) High parasternal or subcostal views
- 2) Difficult to image

2. Rastelli procedure

- a. Valved conduit from RV to PA; LV to aorta via VSD and intraventricular patch
- b. Increases pulmonary flow; May reestablish proper sequence of flow to PA and aorta
 - 1) TGA + VSD + subvalve PS
 - 2) Truncus arteriosus
 - 3) Double outlet RV
 - 4) Pulmonary atresia with VSD (Tetralogy with pulmonary atresia)

3. Damus-Kaye Stansel

- a. Supravalve end-to-side anastomosis of PA to aorta and valved conduit between RV and main PA
- b. Serves as aorto-pulmonary window to relieve irreparable subvalvular stenosis.

4. Norwood procedure

- a. PA anastomosis to aorta; Conduit from aorta to MPA
- b. Improves and stabilizes flow to aorta
 - 1) Aortic valve atresia
 - 2) Hypoplastic left heart
- c. Staged procedure leading to Fontan operation

Section 12: Cardiac Masses

I. Infectious Masses (see Section 7, XI)

- A. Vegetation due to infective endocarditis
- B. Noninfectious vegetations (Nonbacterial thrombotic endocarditi(s))

II. Thrombi

A. Left ventricular thrombus

1. Predisposing conditions (**underlying wall motion abnormality**)
 - a. Apical akinesis (especially acute anterior MI)
 - b. LV aneurysm
 - c. Diffuse left ventricular systolic dysfunction
2. Echocardiographic features
 - a. Contour distinct from endocardial border
 - b. Often (but not always) more echogenic than underlying myocardium
 - c. Often (but not always) convex surface
 - d. Located in region of abnormal wall motion
3. Technical suggestions (scanning techniques)
 - a. Use high-frequency, short-focus transducers
 - b. Decrease depth of field
 - c. Move focal zone nearer apex
 - d. Use low transmit power and gain
 - e. Addition of non-standard views
 - f. Contrast enhancement occasionally helpful
4. Pitfalls
 - a. Near-field and ring-down artifact
 - b. Prominent trabeculations
 - c. Papillary muscles
 - d. False tendons
 - e. Layered (non-protruding) thrombi more difficult to appreciate
 - f. New thrombi are less echogenic

5. Potential for embolization

- a. Size
- b. Protrusion into cavity
- c. Mobility

B. Left atrial thrombus

1. Predisposing factors

- a. Atrial fibrillation
- b. Mitral stenosis
- c. Prosthetic mitral valve
- d. Left atrial enlargement
- e. Low cardiac output

2. Association with spontaneous echo contrast

3. Location

- a. Left atrium
- b. Left atrial appendage (poorly imaged with TTE)

4. TEE more sensitive than TTE

5. Clinical significance

- a. Increased risk of thromboembolism
- b. Relative contraindication to balloon mitral valvotomy

C. Right atrial thrombi

1. Predisposing factors

- a. Atrial fibrillation
- b. Right atrial enlargement
- c. Catheters, pacemakers

2. Clinical significance

- a. Embolization (pulmonary or paradoxical)
- b. Thrombi on catheters, pacemakers have potential for infection

3. RA emboli-in-transit

4. Need to distinguish thrombi from:
 - a. Congenital remnants (Eustachian valve; Chiari network)
 - b. Microbubbles
 - c. Reverberation artifacts
 - d. Lipomatous hypertrophy of atrial septum

D. Right ventricular thrombi relatively uncommon

III. Cardiac Tumors

A. Primary

1. Benign

- a. Myxoma
- b. Papillary fibroelastoma
- c. Lipoma
- d. Fibroma
- e. Hemangioma
- f. Miscellaneous others

2. Malignant

- a. Sarcomas
 - 1) Angiosarcoma
 - 2) Rhabdomyosarcoma
 - 3) Fibrosarcoma
 - 4) Extraskeletal osteosarcoma
- b. Mesothelioma
- c. Malignant lymphoma
- d. Miscellaneous others

B. Secondary (Metastatic)

1. Most often metastasize to visceral pericardium
2. Origin of metastatic tumors in adults (order of frequency)
 - a. Lung
 - b. Lymphoma
 - c. Breast

- d. Leukemia
- e. Stomach
- f. Melanoma
- g. Liver
- h. Colon

3. Tumors invading right heart, via IVC

- a. Renal cell carcinoma (Hypernephoma)
- b. Hepatocellular
- c. Uterine tumors

C. Role of echocardiography for evaluating cardiac tumors

1. Detection and characterization

a. Morphology

- 1) Size
- 2) Shape
- 3) Borders
- 4) Deformability
- 5) Homogeneity

- b. Location, single, multiple
- c. Site and nature of attachment
- d. Infiltration (suggests malignant)

2. Differential diagnosis

3. Guidance of biopsy, surgery

4. Limitations

- a. Inability to determine histology
- b. Limited acoustic access in some patients
- c. Limited “field of view” (mediastinum, adjacent structure)

IV. Miscellaneous Non-Neoplastic Intracardiac Masses

A. Blood cyst

B.

V. Extracardiac “masses”

A. Cysts

1. Pericardial
2. Bronchogenic

B. Mediastinal tumors

C. Aorta

1. Tortuous
2. Aneurysms

VI. Structures mistaken for abnormal cardiac mass

A. Left atrium

1. Ridge between left superior pulmonary vein and LA-appendage
2. Atrial suture line after cardiac transplant
3. Inverted LA-appendage
4. Atrial septal aneurysm
5. Lipomatous hypertrophy of atrial septum
6. Pectinate muscles in LA-appendage
7. Tortuous descending thoracic aorta “compressing” LA

B. Right atrium

1. Crista terminalis
2. Congenital remnants (Eustachian valve, Chiari network)
3. Lipomatous hypertrophy of atrial septum
4. Trabeculations of right atrial appendage
5. Atrial suture line after cardiac transplant
6. Catheters, central venous lines, pacemaker wires

C. Left ventricle

1. Papillary muscles
2. Anomalous bands (false tendons)
3. Prominent muscular trabeculations
4. Prominent mitral annular calcification

D. Right ventricle

1. Moderator band
2. Papillary muscles
3. Catheter or pacemaker wire

E. Aortic valve

1. Nodules of Arantius
2. Lambl's excrescences
3. Aortic cusp imaged *en face* in diastole (TEE)

F. Mitral valve

1. Redundant chordae
2. Myxomatous mitral valve tissue

G. Pericardium

1. Epicardial adipose tissue
2. Fibrinous debris in a chronic pericardial effusion

Section 13. Diseases of the Aorta

I. Aortic Dissection

A. Echo findings

1. Visualization of dissection flap
2. Dilated aorta
3. Widening of aortic wall (intramural hematoma)
4. Aortic insufficiency
5. Pericardial and/or pleural effusion
6. Compression of left atrium

B. Goals of imaging

1. Confirmation or exclusion of diagnosis
2. Determination of location (ie Type A or B)
3. Determination of extent of dissection
4. Presence, severity, and mechanism of aortic insufficiency
5. Presence of pericardial and/or pleural effusion
6. Involvement of coronary arteries
7. Involvement of major branch vessels
8. Detection of rupture
9. Less important features
 - a. Localization of intimal tear (entry site)
 - b. Detection of re-entry site(s)
 - c. Flow dynamics with true and false lumens

C. Transesophageal echocardiography

1. Sensitivity, specificity
2. Advantages
3. Disadvantages
4. Superiority over TTE
5. Comparison with other imaging modalities
 - a. Aortography
 - b. CT-scan (including fast spiral CT)
 - c. MRI

6. Pitfalls

- a. Reverberations, catheters
- b. Mirror-image artifacts
- c. Spiral flow down descending aorta
- d. Hemiazygous sheath
- e. Thoracic aortic aneurysm with mural thrombus
- f. “Blind spot” (can miss type II dissection)

D. Intramural hematoma (“Atypical” aortic dissection)

1. Pathogenesis

- a. Small intimal tears
- b. Ruptured vasa vasorum
- c. Penetrating ulcer
- d. Trauma

2. Prevalence (10-15% of aortic dissections)

3. Echo findings

- a. Crescentic or circumferential thickening of aortic wall
- b. Absence of dissection flap
- c. Preserved aortic lumen

4. Clinical significance

5. Differential diagnosis

- a. Aortic aneurysm with mural thrombus
- b. Hemiazygous sheath
- c. Atherosclerotic thickening of aortic wall

E. Natural history, fate of false lumen, post-op complications

1. False lumen remains patent approximately 80%

2. Post surgical complications of aortic dissection

- a. Aneurysmal dilatation of false lumen
- b. Subsequent aortic rupture
- c. Re-dissection
- d. Pseudoaneurysm at anastomotic sites
- e. Progressive aortic insufficiency

F. Mechanisms of aortic insufficiency

1. Dilatation of aortic root
2. Asymmetric dissection causes faulty coaptation
3. Dissection into commissure with faulty coaptation
4. Dissection flap prolapses through aortic valve

II. Thoracic Aortic Aneurysms

A. Definition: True aortic aneurysms are dilatations of the aorta that contain all 3 layers (intima, media, adventitia)

B. Pathophysiology: caused by weakening of the media

C. Classification

1. Morphologic

- a. Fusiform
- b. Saccular

2. Location

- a. Ascending aorta (45%)
- b. Aortic arch (10%)
- c. Descending thoracic aorta (35%)
- d. Thoraco-abdominal aorta (10%)

D. Etiology

1. Atherosclerosis
2. Medial degeneration
 - a. Idiopathic (annuloaortic ectasia)
 - b. Marfan's syndrome
 - c. Other heritable disorders
 - d. Associated with bicuspid aortic valve
3. Aortic dissection with dilatation of persisting false lumen
4. Trauma with incomplete aortic rupture
5. Syphilis
6. Mycotic (bacterial, fungal, tuberculous aortitis)
7. Non-infectious aortitis (Giant-cell, Takayasu's)

E. Goals of imaging thoracic aortic aneurysms

1. Confirm or exclude diagnosis
2. Measure maximal diameter
3. Define longitudinal extent
4. Determine involvement of aortic valve
5. Determine involvement of arch vessel(s)
6. Detect periaortic hematoma or other signs of leakage
7. Differentiate from aortic dissection
8. Detect mural thrombus

F. Echo characteristics

G. Role of TEE

H. Differentiation from aortic dissection with thrombosed false lumen

I. Comparison of TEE with other imaging modalities

1. TEE probably equivalent to CT-scan, MRI, aortography (paucity of data)
2. Each modality has strengths/limitation

J. Complications

1. Aortic insufficiency
2. Rupture
 - a. Pericardium
 - b. Left pleural space
3. Aortic dissection
4. Thromboembolism
5. Compression of adjacent mediastinal structures
6. Fistula formation

K. When to operate on ascending aortic aneurysms

1. Controversial
2. Depends on many factors:
 - a. Etiology of aortic root disease (eg. tendency to operate sooner in Marfan syndrome)
 - b. Size
 - c. Rate of change of size
 - d. Skill, experience of surgeon
3. General guidelines

III. Traumatic Injury of the Aorta

A. Wide spectrum of extent of injury/pathology

1. Simple contusion
2. Intimal tear
3. Intramural hematoma
4. False aneurysm
5. Frank rupture
6. Major dissection **not** a feature of aortic trauma (usually no underlying medial disease)

B. Intimal lacerations

1. Majority are horizontal
2. May be small, limited or large, circumferential
3. Develop from within, extend outward
 - a. Intima only
 - b. Intima and varying amounts of media
 - c. Full thickness of aortic wall
 - d. Adventitia is toughest layer

C. Role of echo (TEE)

1. Diagnosis
2. Location and extent of aortic disruption
3. Associated complications

D. Echo findings

1. Intimal tears
 - a. Thin, mobile intraluminal appendages
 - b. Usually in vicinity of isthmus
 - c. Small and superficial
 - d. Diameter and contour of aorta unchanged
 - e. No turbulence by color Doppler
 - f. Thrombus may develop at site of small tear
2. Intramural hematoma

E. Diagnostic accuracy

F. Comparison of TEE with other imaging modalities (pros and cons)

1. Aortography
2. CT-scan
3. MRI

IV. Aortic Atherosclerosis

A. Transesophageal echo

1. Detection
2. Grading
 - a. Size
 - b. Mobility
 - c. Ulceration

B. Clinical relevance

1. Risk of embolization
2. Marker for CAD, carotid artery disease, PVD

C. Management issues

D. Role of epiaortic imaging in OR (see Section 17, II, B)

E. Natural history of aortic atherosclerosis

V. Sinus of Valsalva Aneurysms

A. Location

1. Right coronary sinus (70%); project into RV>RA
2. Noncoronary sinus (25%); project into RA
3. Left coronary sinus (5%); project into LA>LV

B. Role of Echo

1. Detection (conventional TEE detects 75%)
2. Delineate location, size, shape of aneurysmal sac
3. Localize sites of rupture
4. Identify presence/absence associated abnormalities
5. Detect and assess complications
 - a. Rupture into cardiac chamber
 - b. RV outflow tract obstruction
 - c. Endocarditis

- d. Aortic regurgitation
- e. Tricuspid regurgitation
- f. Erosion into ventricular septum
- g. Obstruction of adjacent structures
 - 1) Coronary artery compression
 - 2) SVC obstruction
 - 3) Tricuspid stenosis

- 6. Differentiate from other causes of continuous murmur
- 7. Serial evaluation of unruptured aneurysm

C. Echo features

1. M-mode

- a. Fluttering of tricuspid valve
- b. Early closure of anterior cusp of aortic valve
- c. Premature opening of pulmonic valve
- d. Right ventricular volume overload

2. 2D-echo

- a. Round or fingerlike (windsock) outpouchings
- b. Size and shape may change during cardiac cycle

3. Doppler

- a. Continuous, high velocity, turbulent flow
- b. Typically into RV or RA
- c. May be difficult in presence of VSD

Section 14 Stress Echocardiography

I. Basic Principles

- A. Determinants of myocardial oxygen demand
- B. Ischemic cascade (sequence of events in ischemia)
- C. Coronary flow reserve
- D. Relationship between coronary artery anatomy and segmental wall motion

II. Technical and Interpretative Aspects

- A. Echo views for evaluation of LV wall motion
- B. Types of exercise (pros and cons)

- 1. Treadmill
- 2. Bicycle
 - a. Upright
 - b. Supine
- 3. Pacing
- 4. Miscellaneous
 - a. Handgrip
 - b. Cold presser
 - c. Mental stress

C. Pharmacologic

- 1. Dobutamine
- 2. Arbutamine
- 3. Dipyridamole
- 4. Adenosine

D. End-points

- 1. Target heart rate
- 2. Clinical ischemia (intolerable chest pain)
- 3. Hypotension (BP <90 or fall in systolic pressure >30 mmHg from baseline)
- 4. Hypertension (BP \geq 220 mmHg)
- 5. Sustained V-tach or SVT
- 6. Obvious new WMA involving \geq 2 segments

E. Relative contraindications

1. Unstable angina
2. Uncontrolled hypertension
3. Serious, uncontrolled arrhythmias
4. Mobile LV thrombus
5. Severe aortic stenosis
6. Hypertrophic obstructive cardiomyopathy
7. Decompensated CHF

F. Wall motion score index

1. Evaluate pre and post-stress

G. Interpretation

1. Bayesian analysis
2. Criteria for positive test
3. Interobserver variability
4. Reproducibility
5. Causes of false positive tests
 - a. Nonischemic cardiomyopathy
 - b. Septal motion abnormalities (LBBB, s/p CABG)
 - c. Basal inferior wall
 - d. Excessive hypertensive response
 - e. Poor image quality
 - f. Interpreter bias
6. Causes of false negative tests
 - a. Single vessel disease
 - b. "Mild" coronary stenosis
 - c. Left circumflex artery
 - d. Inadequate stress
 - e. Rapid recovery
 - f. Poor image quality

H. Limitations

1. Image quality
2. Respiratory interference
3. Translation, rotation
4. Baseline abnormal global or regional function

- 5. Arrhythmias
- I. Safety/complications

III. Accuracy

A. Sensitivity and specificity

- 1. In general population
- 2. In coronary disease population
- 3. By individual vessels
- 4. Single vs multivessel disease

B. Comparisons

- 1. With exercise ECG
- 2. With nuclear tests
- 3. Stress echo
 - a. Treadmill vs bicycle
 - b. Exercise vs pharmacologic
 - c. Comparison of various pharmacologic agents

IV. Dobutamine Stress Echo

A. Basic principles

B. Special topics

- 1. Role of atropine
- 2. Hypotension
- 3. LV outflow tract obstruction
- 4. "Optimal" infusion protocol
 - a. Begin with 5 mg or 10 mg?
 - b. Stages: 3 minutes vs 5 minutes
 - c. Peak dose: 40 vs 50 mcg/kg/min

V. Prognostic Role of Stress Echo

A. Various populations

- 1. General referral
- 2. Known CAD
- 3. Post myocardial infarction
- 4. Post revascularization (thrombolysis, PTCA, CABG)

- B. Pre-op evaluation prior to noncardiac surgery

VI. Myocardial Viability

A. Basic principles

1. Myocardial stunning
2. Hibernating myocardium

B. Dobutamine stress echo

1. Interpretation
 - a. Biphasic response
 - 1) Viable myocardium
 - 2) Predicts recovery of function
 - b. Improvement at both low and high dose
 - 1) Viable myocardium
 - 2) Less predictive or recovery function
 - c. No improvement
 - 1) Myocardial infarct (scar)
 - 2) Non-viable myocardium
2. Accuracy
3. Comparison with other tests (post-revascularization recovery of function)
 - a. PET
 - b. Thallium

VII. TEE-Stress Echo

- A. Pharmacologic
- B. Pacing

VIII. Stress Echo for Hemodynamics and Valve Disease

- A. Aortic stenosis
- B. Mitral stenosis
- C. Valve regurgitation
- D. Prosthetic valves
- E. Pulmonary hypertension
- F. Hypertrophic cardiomyopathy
- G. Diastolic function

XI. Special Topics

- A. Stress echo in women
- B. Beta blockers
- C. LVH, LBBB
- D. Transplant CAD

Section 15: Transesophageal Echocardiography

I. The Procedure

- A. Historical perspective
- B. Laboratory setup
 - 1. Equipment, supplies
 - 2. Medications
 - 3. Patient preparation
- C. Technique
 - 1. Probe insertion
 - 2. Probe manipulation
- D. Relative contraindications
 - 1. Pre-existing esophageal pathology
 - 2. Esophageal diverticulum
 - 3. Esophageal varices
 - 4. Recent esophageal surgery
 - 5. Active UGI bleed
 - 6. Perforated viscus (known or suspected)
 - 7. Severe cervical arthritis
 - 8. Profound oropharyngeal distortion
 - 9. Unwilling or uncooperative patient
- E. Complications
 - 1. Arrhythmias
 - 2. Respiratory distress, hypoxia
 - 3. Transient hypo or hypertension
 - 4. Aspiration
 - 5. Laryngospasm, bronchospasm
 - 6. Perforation of hypopharynx, esophagus
 - 7. Laryngeal nerve damage
- F. Anatomic imaging views

II. Clinical Applications

- A. Infective endocarditis
- B. Cardiac sources of embolism
 - 1. Left atrial thrombi
 - 2. Left ventricular thrombi
 - 3. Prosthetic valve thrombi
 - 4. Valve “strands”
 - 5. Vegetations
 - 6. Intracardiac tumors
 - 7. Thoracic aortic atherosclerosis
 - 8. Patent foramen ovale
 - 9. Atrial septal aneurysms
 - 10. Left atrial spontaneous echo contrast
 - 11. Mitral annular calcification
 - 12. Mitral valve prolapse
- C. Native valve disease
- D. Prosthetic valve disease
- E. Thoracic aortic pathology
 - 1. Aortic dissection
 - 2. Aortic aneurysm
 - 3. Aortic trauma
 - 4. Aortic atherosclerosis
- F. Intracardiac masses
- G. Coronary arteries
- H. Congenital heart disease
- I. Critically ill patients
- J. Intraoperative applications
- K. Guidance of interventional procedures
- L. Stress echo using TEE

Section 16: Contrast Echocardiography & Tissue Harmonic Imaging

1. Bubble Physics, Pharmacology, Safety

A. Bubble characteristics

1. Size
2. Stability (microbubble persistence)
 - a. Radius
 - b. Gas density
 - c. Diffusivity
 - d. Gas composition
 - e. Encapsulation (shell, surface characteristics)
 - f. Saturation concentration
3. Resonant frequency

B. Acoustic properties of microbubbles

1. Different acoustic impedance than blood
2. Intensity of reflections independent of direction of sound source
3. High ultrasonic backscatter

C. External influences on contrast agents

1. Ambient pressure
2. Acoustic pressure

D. Safety

II. Contrast Agents

A. Ideal contrast agent

1. Nontoxic (complete safety)
2. Inert and poorly soluble gas
3. Small size (for transcapillary passage)
4. Excellent opacification (reflectivity)
5. Capable of oscillation upon ultrasonic stimulation (to allow detection of harmonic images)
6. Long half-life
7. Intravenous administration
8. Similar rheology to RBCs

B. Early hand-agitated agents

1. Agents

- a. Normal saline
- b. Indocyanine green
- c. Carbon dioxide
- d. Hydrogen peroxide
- e. Blood

2. Limitations

- a. Short, variable half-life
- b. Large, variable size
- c. No transpulmonary circulation

C. Early sonicated agents

1. Agents

- a. Dextrose
- b. Renograffin
- c. Albumen

2. Limitations

- a. Short, variable half-life
- b. Large, variable size

D. Properties of newer contrast agents

1. Outer shell

- a. Phospholipid
- b. Saccharides
- c. Albumen
- d. Biopolymers

2. Non-diffusible gases

- a. Perfluorocarbons
- b. Sulfur hexafluoride

3. Phase-shift technology

4. Decreased acoustic destruction

5. Transpulmonary circulation

III. Imaging Instrumentation for Contrast Agents

A. Imaging modalities for contrast detection

1. Image mode
 - a. Fundamental mode
 - b. Harmonic imaging
 - c. Color Doppler (fundamental)
 - d. Integrated backscatter
 - e. Power Doppler imaging
 - f. Pulse inversion imaging
2. Capture mode
 - a. Continuous
 - b. Triggered (intermittant; gated)
 - c. Destruction/fill imaging
 - d. Sequential pulse imaging
3. Analysis mode
 - a. Visual raw image
 - b. Color coding
 - c. Backing round subtraction
 - d. Quantitation (densitometry)
 - e. Cyclic variation

B. Instrumentation issues

1. Wide dynamic range
2. Narrow transmit spectrum
3. Sharp receiver filter

IV. Clinical Applications (Practical, Theoretical, Experimental)

A. Endocardial border enhancement

1. Global and regional wall motion evaluation
2. Determination of volume, ejection fraction
3. Stress echocardiography

B. Doppler signal enhancement

1. Aortic stenosis
2. TR jet/pulmonary artery pressure
3. Mitral regurgitation
4. Pulmonary venous flow

C. Characterization of intracardiac shunts and flow patterns

D. Cardiac output

1. Application of indicator dilution therapy to contrast appearance/delay
2. Too many variables to be practical

E. Aortic dissection (Highlight flow differential in true/false lumens)

F. Myocardial perfusion (Echo Lab and Cath Lab)

1. Coronary physiology

- a. Relative vascular volume
- b. Perfusion velocity
- c. Relative regional perfusion

2. Hypofusion/ischemia (rest and stress)
3. Quantification of infarct size
4. Delineation of area(s) “at risk” within coronary territories
5. Delineation of collateral flow (area of myocardium opacified by 2 separate injections: one into right and one into left coronary artery)
6. Determination of myocardial viability
7. Assessment of coronary flow reserve
8. Assessment of success of reperfusion (angioplasty)
9. Document “no-reflow” phenomenon after reperfusion

- a. Expression of microcirculatory damage
- b. Predictor of poor regional contractile function

G. Myocardial perfusion (Operating room)

1. Determine distribution and adequacy of cardioplegia delivery
 - a. If underperfused, consider retrograde cardioplegia
 - b. Sequence of graft placement (underperfused area first)
2. Determine success of graft placement

V. Technical Aspects of Myocardial Perfusion

A. Quantitation

1. Videointensity
2. Radiofrequency imaging

B. Limitations

1. Inadequate quantitation of coronary flow
2. Limited data on reproducibility
3. Safety issues (intracoronary)
4. "Ideal" dosing (bolus vs infusion)
5. Attenuation artifacts

C. Areas of development

1. New triggering algorithms
 - a. 1:1, 1:2, 1:4, 1:8,.....
 - b. End-diastole vs end-systole
 - c. Accelerated intermittent imaging
(low frame rate, low mechanical index)
 - d. Burst imaging
2. Digital processing
 - a. Background subtraction
 - b. Correction for translation
 - c. Color coding
 - d. Quantification of pixel intensity in multiple ROIs

VI. Future Developments (Contrast)

- A. Novel site-targeted contrast agents (local gene or drug therapy)
- B. Enhance therapeutics
 1. Accelerate thrombolysis
 2. Ultrasound angioplasty
- C. Continued development of IV contrast agents to enhance myocardial perfusion
- D. Modification of ultrasound equipment for contrast use

VII. Harmonic Imaging

A. Principles of harmonic imaging

1. Definition: Transducer emits a given frequency and receives at multiples of that frequency
2. Principles of harmonic imaging
 - a. Fundamental frequency
 - b. Harmonic frequencies
3. Methods of amplifying selective frequencies
 - a. Harmonic imaging
 - b. Frequency conversion technology
 - c. Fusion technology

B. Impact on image quality

C. Use in contrast echocardiography

D. Use in endocardial edge definition

E. Adjunctive techniques

1. Triggered imaging (transient response imaging; intermittent imaging)
2. Power Doppler imaging (Power “angio”, “energy” mode, loss of correlation)
3. Dual triggering

Section 17: Intraoperative Echocardiography

1. Echocardiographic Approaches

- A. Epicardial echo (2D and Doppler)
- B. TEE
- C. Strengths/weaknesses of each

II. Established Indications for Intraoperative Echo

A. Guidance and assessment of surgical procedures

1. Valve repair

- a. Mitral (see Section 7 – G)
- b. Aortic
- c. Tricuspid

2. Infective endocarditis

- a. Delineation of valve damage
- b. Detection of complications
 - 1) Abscesses
 - 2) Fistulae
 - 3) Aorto-LV discontinuity
- c. Indications and methods of repair

3. Prosthetic valves

- a. Normal seating
- b. Supra-annular insertion
- c. Physiologic regurgitation
 - 1) Central
 - 2) “Stitch” leaks

d. Complications

- 1) Pathologic regurgitation
- 2) Myocardial rupture
- 3) Coronary sinus ligation
- 4) Circumflex artery ligation
- 5) Retained mitral apparatus interfering with prosthetic function
- 6) Stuck disc(s)

4. Aortic surgery

a. Aortic dissection

- 1) Confirmation of diagnosis
- 2) Site and extension (critical: is ascending aorta involved?)
- 3) Presence, mechanism, severity of aortic regurgitation
- 4) Involvement of coronary arteries
- 5) Pericardial effusion

b. Aortic aneurysm

- 1) Site
- 2) Extent
- 3) Size
- 4) Rupture

5. Hypertrophic cardiomyopathy (myotomy/myectomy)

a. Define site, extent of resection

- 1) Accurate est'n of septal thickness (depth, length, width)
- 2) Distance from aortic annulus

b. Assess immediate results

- 1) Thinning of septum, widening of LVOT
- 2) Resolution of SAM
- 3) Elimination, reduction gradient
- 4) Elimination, reduction of MR

- c. Detect, exclude complications
 - 1) Septal perforation
 - 2) Worsening MR
 - 3) If muscle bridge unroofed:
 - a. Snaring and occlusion of LAD
 - b. Inadvertant RV perforation
 - 6. Congenital heart disease (see also Section 11, XII)
 - a. Road map for surgeon
 - b. Detection of significant residual defects
 - c. Complications
 - 7. Intracardiac masses
 - a. Attachment site(s)
 - b. Size and morphology
 - c. Relation to adjacent structures
 - 8. Constrictive pericarditis
 - a. Detection of thickened pericardium
 - b. See also Section
 - c. Cardiac function after pericardiectomy
 - 9. Cardiac tamponade
 - a. Size, location
 - b. Loculated or circumferential
 - c. Hematoma
- B. Detection, location, characterization of atherosclerosis
- 1. Type of echo
 - a. TEE
 - b. Epiaortic imaging
 - 1) Imaging planes
 - 2) Sterility
 - 2. Impact on surgery

C. Monitoring of LV function

1. Global function

- a. Ventricular size, volume
- b. Ejection fraction
- c. Cardiac output, stroke volume

2. Segmental function (esp. for myocardial ischemia)

a. Anatomy of segments and coronary distribution

- 1) Transgastric short and long axes
- 2) Mid-esophageal four and 2-chamber views
- 3) Limitations

b. Which patients appropriate (“High-risk”)

1) “High-risk”

- a) Elderly
 - b) Recent myocardial infarct (<3 mos.)
 - c) Unstable angina
 - d) Multiple CAD risk factors
 - e) Severe LV dysfunction
- 2) Major thoracic (noncardiac) or abdominal surgery (especially in patients with poor EF)
 - 3) Major vascular surgery
 - 4) Abdominal aneurysm resection
 - 5) Known CAD patient undergoing emergency noncardiac surgery
 - 6) Mid-CAB surgery

3. Loading conditions (filling pressures) see also Section 3

- a. Chamber size, volume
- b. Mitral inflow
- c. Pulmonary venous flow

D. Detection of intracardiac air

E. Placement and function of assist devices

F. Guidance of minimally invasive surgery

1. Pre-pump

- a. Usual evaluation of LVF, identification of cardiac pathology, etc.

- b. Guidance of placement of devices
 - 1) Vena caval cannula (deep transgastric longitudinal views)
 - 2) Coronary sinus catheter (mid-esophageal tranverse 4-chamber view)
 - 3) PA vent (mid-esophageal transverse short axis of aorta)
 - 4) Endoaortic balloon clamp (mid-esophageal long axis 130° view of aorta)

- c. Identification of intraluminal obstruction (atheroma, thrombi)
- d. Identification of anatomical variants (chamber dilatation, persistent left SVC)

2. On-pump

- a. Keep devices in proper location (esp. endoaortic clamp)

3. Post-pump
 - a. Monitoring for evacuation of air
 - b. Intravascular volume determination
 - c. Identification of ischemia (new WMA)
 - d. Usual evaluation of success of procedure
 - e. (eg. persistent regurgitation)

III. Less Well Established Indications for Intraop Echo

A. Supported in weaker evidence and expert consensus

1. Monitoring patients with increased risk of myocardial ischemia
2. Monitoring patients with increased risk of hemodynamics disturbances
3. Assessment and repair of cardiac aneurysm
4. Evaluation and removal of foreign bodies
5. Detection of foreign bodies
6. Intracardiac thrombectomy
7. Pulmonary embolectomy
8. Suspected cardiac trauma
9. Repair of aortic dissection without aortic valve involvement
10. Evaluation of pericardial surgery
11. Evaluation of anastomotic sites during heart and lung transplant

B. Little current scientific or expert support

1. Evaluation of myocardial perfusion, coronary anatomy, graft patency
2. Repair of nonhypertrophic cardiomyopathies
3. Monitoring cardioplegia administration
4. Monitoring for emboli during orthopedic procedures
5. Assessment of repair of thoracic aortic pathology
6. Monitoring air in left ventricle

IV. Technical consideration

A. Appliances used during cardiopulmonary bypass

1. Mediastinal/intracardiac catheters
2. Vents
3. Arterial, venous, and coronary sinus cannulae
4. Vascular clamps

B. Effects of anesthesia and surgery on cardiac physiology

1. **Prior to** cardiopulmonary bypass

- a. Controlled ventilation
- b. Anesthetic drugs
- c. Non-anesthetic drugs commonly used during surgery
- d. Arterial cannulation
 - 1) Atheroma
 - 2) Retrograde aortic flow with femoral artery cannulation
 - 3) Aortic dissection
- e. Venous cannulation
 - 1) Hypertension in SVC
 - 2) Atrial compliance
 - 3) Arrhythmias

2. **During** cardiopulmonary bypass

- a. Hyperthermia
- b. Hemodilution
- c. Nonpulsatile flow
- d. Circulatory arrest
- e. Intracavitary air
- f. Myocardial perfusion
 - 1) Antegrade delivery
 - 2) Retrograde delivery
 - 3) LV dilatation associated with aortic regurgitation
- g. Cardioplegia
- h. Spontaneous echo contrast

3. During **weaning** from cardiopulmonary bypass

- a. Assessment of LV function
 - 1) Global and regional
 - 2) Ejection fraction
 - 3) Fractional area change
 - 4) Mitral inflow pattern
 - 5) Pulmonary vein pattern

6) Influence of preload and afterload changes

- b. Assessment of RV function
- c. Assessment of diastolic function
- d. Air detection

4. **After** cardiopulmonary bypass

- a. Protamine
- b. LV and RV contractile function
- c. Volume status (preload)

Section 18: Interventional Echocardiography

I. Role of Echo for Guidance of Interventional Procedure

- A. Intraoperative echocardiography (see Section 17)
- B. Echo-guided pericardiocentesis
 - 1. Therapy of choice for most pericardial drainages
 - 2. Simple, safe, effective
 - 3. Higher incidence of recurrence than surgical drainage ?
 - 4. Complications
 - a. Chamber puncture
 - b. Vessel injury
 - c. Pneumothorax
 - d. Infection
 - e. Ventricular arrhythmias
- C. Guide endomyocardial biopsy
- D. Guide balloon and blade atrial septostomy
- E. Guide catheter-based closure of ASD and VSD
- F. Transvenous “rescue” of intracardiac foreign bodies
- G. Monitor high-risk complex coronary interventions in the cardiac cath lab
- H. Guide catheter ablation of cardiac arrhythmias
- I. Guide percutaneous balloon valvotomy
- J. Guide surgical or percutaneous transmyocardial laser revascularization

II. Intravascular Ultrasound

- A. Introduction
 - 1. Provides tomographic images of coronary arteries and blood vessels
 - 2. An evolving technology
- B. Technical aspects: Transducers (2 types)
 - 1. Mechanical
 - a. Simple design: single rotating imaging crystal
 - b. Larger acoustic aperture: slightly better penetration
 - c. Minimal dead zone (mirror)
 - d. Speed \approx 1600-1800 rpm
 - e. Drive shaft/nonuniform rotational distortion (disadvantage)

2. Phased array (multiple element array)
 - a. Consecutive activation of multiple elements (complex computing)
 - b. More flexible
 - c. Suitable for mating to balloon and atherectomy catheters
 - d. Larger dead zone
 - e. Small effective aperture

3. General
 - a. Tsdrs typically 10-40 MHz
 - b. Cath diameter
 - 1) 2.9 – 3.5 Fr (coronaries)
 - 2) 4 – 10 Fr (larger peripheral vessels)
 - c. Axial resolution 120 – 300 microns

C. Technical aspects

1. Guidewire configuration
 - a. Over-the-wire
 - b. Monorail
 - c. Fixed guide-wire tip

2. Catheter preparation
 - a. Mechanical (flushing)
 - b. Phased array (ring-down)

3. Imaging protocol
 - a. Slow (often motorized) pullback
 - b. Distal to proximal

4. Imaging landmarks and coronary anatomy

D. IVUS characteristics of arteries

1. Normal intima – hyperechoic
 - a. Endothelial cell layer
 - b. Internal elastic membrane

- c. ≤ 2 mm thick
- 2. Media – relatively anechoic
 - a. Circumferential oriented smooth muscle cells
 - b. Matrix, collagen, elastin
- 3. Adventitia – hyperechoic
 - a. Collagen
 - b. Elastic fibers
- 4. Perivascular structure and/landmarks (veins, pericardium, myocardium)
- 5. Normal range of coronary artery dimensions (mean)

a. Left main	2.5 to 5.5 mm (4.0 mm)
b. LAD	2.0 to 5.0 mm (3.6 mm)
c. Prox LCx	1.5 to 5.0 mm (3.0 mm)
d. RCA	1.5 to 5.0 mm (3.2 mm)

E. IVUS characteristics of veins

1. Thin, hyperechoic walls
2. Lack distinct layers
3. Shape dependent on transmural distending pressures

F. IVUS characteristics of atherosclerotic plaque

1. Composition (qualitative descriptions)
 - a. Calcium
 - 1) Intensely echo-reflective (white)
 - 2) Acoustic shadowing
 - b. Fibrous tissue
 - 1) Moderately echo-reflective (grey)
 - 2) Echo reflectivity < than adventitia
 - 3) May be fibromuscular
 - 4) Can also be dense fibrous tissue (echo reflectivity \geq adventitia)
 - c. Lipid
 - 1) Relatively anechoic (black)
 - 2) Fibro-fatty plaque hypoechoic

- d. Mixed (can be multiple plaque components)
2. Distribution

- a. Concentric vs eccentric
- b. Longitudinal distribution

3. Plaque burden

- a. Minimal $\leq 20\%$ of VA occupied by plaque
 - b. Moderate $>20\% \leq 40\%$ of VA occupied by plaque
 - c. Large $>40\% \leq 60\%$ of VA occupied by plaque
 - d. Massive $>60\%$ of VA occupied by plaque
- VA = total vessel area

4. Wall disruptions

- a. Rupture radial tear, perpendicular to vessel wall
- b. Dissection longitudinal tear, parallel to vessel wall

- 1) Visualization of blood flow in newly created lumen
- 2) May be confirmed by saline or contrast injection
- 3) May see pulsatility of echolucent area within or behind plaque

c. Characteristics to be specified

- 1) Location proximal, distal, at target stenosis
- 2) Axial length in mm
- 3) Circumferential extension arc in hours or degrees

d. Maximal depth

- 1) Partial plaque b/w tear and adventitia
- 2) Complete full thickness tear extending through the plaque to the adventitia

e. More readily appreciated when matched to pre-interventional images

G. Other structures

1. Blood speckle
2. Thrombus
3. Aneurysm/pseudoaneurysm
4. Hematoma/intramural hematoma
5. Dissection (simple and complex)

H. Applications of IVUS: Coronaries

1. Diagnostic adjunct

a. Quantitative

- 1) Diameter, circumference, area
- 2) % stenosis

b. Qualitative

- 1) Location of branch vessels/bifurcation
- 2) Plaque morphology
- 3) Plaque vs thrombus

c. Evaluate ambiguous or intermediate angiographic lesions

d. Angiographically normal coronary arteries

- 1) 10-15% pts with suspected CAD
- 2) IVUS demonstrates lesions in 10-15%

2. Guidance of interventional procedure

a. Device selection

- 1) Determined by plaque morphology
- 2) Sizing of PTCA balloons, atherectomy, catheters, stents

b. Assess results

- 1) Quantification of luminal diameter and area
- 2) Detection of residual areas of stenosis (residual plaque burden)

c. Evaluate mechanism(s) of luminal expansion

- 1) Plaque compression or disruption
- 2) Vessel stretching/remodeling

d. Detect presence, location, extent of dissection

- e. Specific devices
 - 1. PTCA: selection of balloon diameter
identification pseudo-successful results
detection of dissection
 - 2. HSRCA: selection of most suitable lesions
(diffuse subendothelial calcium)
 - 3. DCA: exclusion of non-suitable lesions
selection of cut direction
adequacy of plaque removal
identify adventitial cuts
 - 4. STENT: degree of expansion
completeness of apposition
proximal/distal stenoses
(residual plaque burden at edges)
dissections
distribution of restenotic tissue within stent
- 3. Evaluate abrupt closure (thrombosis, dissection)
- 4. Evaluate/facilitate stent deployment
 - a. Adequacy of lesion coverage by stent
 - b. Expansion of stent (complete vs incomplete)
 - c. Apposition to internal vessel wall or compressed plaque
 - d. Lumen size (area) after stent placement (predictor of restenosis)
 - e. Assess intimal hyperplasia after stenting
 - f. Assess for edge dissections
- 5. Detect transplant coronary arteriopathy
 - a. Most important cause mortality/morbidity after 1 yr
 - b. Clinical events occur without prodromal angina
 - c. Distinctive pathology
 - 1) Concentric intimal proliferation
 - 2) Progresses to arterial obliteration
 - 3) Throughout coronary tree (diffuse)
 - d. Earlier detection than angiography
 - e. Mean intimal thickness ≥ 0.3 mm predictor of survival

6. Detect muscle bridging
7. Detect coronary aneurysm/pseudoaneurysm
8. Study vascular biomechanics
9. May provide improved estimates of prognosis
 - a. Abrupt closure
 - b. Death or MI
 - c. Restenosis

I. Applications of IVUS: Great vessels

1. Monitor fenestration of aortic dissection
2. Deployment and evaluation of IVC filters
3. Deployment of endoluminal grafts/stents
 - a. Sizing of proximal & distal neck
 - b. Intraprocedure evaluation of stent struts/hooks
(determine if stent properly expanded)
4. Evaluate pulmonary artery thromboembolic disease

J. Applications IVUS: Therapeutic

1. Clot or plaque pulverization
2. Lithotripsy

K. Limitations of IVUS

1. Requires additional time
2. Catheters are side-viewing (must cross a lesion)
3. Cath not parallel to long-axis of vessel may overestimate xs
area
4. Shadowing and echo-dropout
5. Artifacts
 - a. Ring-down artifact
 - b. Air bubbles
 - c. Ghost images
6. Pitfalls (potential sources of error)
 - a. Catheter malalignment
 - 1) Non-coaxial tsdr alignment
 - 2) Elliptic plane (rather than circular)

- 3) Overestimation of areas/diameters
- 4) Esp. in aorto-ostial lesions in tortuous segments, in large or ectatic vessels

b. Non-uniform rotational distortion (NURD)

- 1) Mechanical imaging catheters
- 2) Measurements may not be reliable

c. Systolic-diastolic changes($\approx 8\%$ pulsatile variability)

- 1) Max area in mid-systole
- 2) Min area in late-diastole
- 3) “Tunnelled” artery \rightarrow systolic compression

L. Limitations of angiography

1. 2D longitudinal projection of 3D-structure
2. Discrepancy b/w angiogr severity & physiologic significance
3. Discrepancy b/w angiography and pathology
4. Discrepancy b/w orthogonal planes
5. “Normal” reference segments often abnormal
6. Magnification makes measurements imprecise
7. Images limited to opacification time
8. Only visualizes, lumen: No information on vessel wall, composition of plaque
9. Underestimates lesion severity in diffuse disease
10. Vessel overlap
11. Ostial stenoses and bifurcation lesions often poorly visualized
12. Extreme obesity, emphysema, chest deformity
13. Requires radiation and iodinated contrast
14. Significant inter-and intraobserver variability

M. 3D-reconstruction

1. Motorized pullback necessary
2. Longitudinal views
3. Longitudinal measurements
4. Rendering

N. Future

1. Forward-looking tsdrs
2. Continued miniaturization, improvements in tsdrs
3. Hybrids of IVUS and interventional catheters (laser, atherectomy, PTCA, etc)

4. Ultrasound guidewires

III. Intracardiac Ultrasonography

A. Applications

- a. Guidance and monitoring of intracardiac interventions
- b. Periop monitoring
- c. Diagnosis of valvular, pericardial, myocardial disease
(when alternative procedures equivocal or impossible)
- d. Assess LV function (from RV catheter)

B. Limitations

- a. Invasive
- b. Depth penetration limited

Section 19: Three-Dimensional Echocardiography

I. Introduction

A. Limitations of 2D-echo

1. Spatial anatomy must be reconstructed mentally
2. Communication of mentally reconstructed images to surgeons not readily performed
3. Lack of depth perception
4. Complex anatomy incompletely visualized
5. Requires assumptions of shape for calculations (eg LV volume, mass)

B. Advantages of 3D-echo

1. Delineate complex orifice shapes
2. Delineate complex chamber, mass, structure shape

II. Methodology

A. Image acquisition

1. Two basic approaches (types of systems)
 - a. 3D-reconstruction
 - 1) On-line acquisition
 - 2) Off-line reconstruction and display
 - b. Real time Volumetric imaging
 - 1) “High-speed” volumetric imaging
 - 2) True real 3D-echo
 - 3) Data acquisition *and* display
2. Localization techniques
 - a. Fixed geometry of image acquisition
 - 1) Parallel planes (“bread-loafing”)
 - 2) Pivot point (rotational or tilt)
 - 3) Mechanically rotating scan heads (multiplane TEE)

- b. Unrestricted scan plane with positional locations
 - 1) Acoustic locator (spark gaps)
 - 2) Electromagnetic locator
- c. Best-fit technique
 - 1) Assumed location of intersecting 2D-planes
 - 2) LIME plot of endocardial surface area
- d. Internal reference systems
 - 1) Fan-like scanning in an arc
 - 2) Rotational scanning
 - 3) Linear (parallel slicing)

B. Image Display

- 1. Wire-mesh (wire-frame)
 - a. Simple, fast
 - b. Lack of anatomic detail
- 2. Tissue-depiction mode (rendering)
 - a. Surface rendering
 - b. Volume rendering

C. Image Processing

- 1. Re-alignment
 - a. Position in space
 - b. Time in cardiac cycle
- 2. Interpolation (filling-in gaps)
- 3. Elimination of artifacts and noise
- 4. Distinction between tissue and blood
- 5. Enhancement of image
 - a. Shading techniques
 - b. Depth encoding
 - c. Lighting techniques
- 6. Selection of region of interest

III. Potential Applications

- A. Ventricular volume, mass, function
- B. Evaluation of mitral valve
 - 1. Mitral valve prolapse
 - 2. Better reconstruction of valve, annulus
 - 3. Mitral valve surgery
- C. Evaluation of aortic valve
 - 1. Number of cusps
 - 2. Eccentric opening
 - 3. Masses
- D. Ventricular reconstructive surgery
 - 1. aneurysm repair
 - 2. Dorr procedure
 - 3. Batista procedure
- E. Complex congenital heart disease
 - a. Diagnosis
 - b. Guide surgical approach
- F. Ischemic heart disease

VI. Limitations

- A. Same as 2D-echo
 - 1. Fundamental physics of ultrasound
 - 2. Dropout, acoustic speckle, beam width
 - 3. Border recognition
- B. Exam time, processing time
- C. Technical
 - 1. Accurate spatial locating system
 - 2. Irregularity of rhythm, respiration
 - 3. Movement of patient and/or probe

Section 20: Miscellaneous Topics

I. Athlete's Heart

II. Systemic Diseases

A. Carcinoid

1. General

- a. Primary tumors: GI tract (>90%), bronchus, ovaries, testicles, pancreas, biliary tract
- b. Carcinoid syndrome in 30%
- c. Cardiac involvement in 50-60% pts w/carcinoid syndrome
- d. Severity cardiac involvement proportional to:
 - 1) Serotonin
 - 2) Kinins
 - 3) Substance P
 - 4) Urinary 5-HIAA

2. Echo findings (predominantly R-sided valve disease)

- a. TV thickening, retraction (97%)
- b. TV may become immobile, fixed in semi-open position
- c. PV cusps thickened, retracted, immobile (\approx 50%)
- d. PS \gg PI
- e. Bivalvular involvement common
- f. RV and RA enlargement (\approx 90%)

3. Left-sided involvement less common

- a. Pts with intracardiac shunt (PFO) or bronchial tumor
- b. Primary carcinoid in pulmonary bronchus
- c. MV thickening (5-10%)
- d. Mod-to-severe MR (<10%)
- e. Aortic valve thickening (<5%)

4. Miscellaneous

- a. Carcinoid metastases to myocardium (<5%)
- b. Small pericardial effusions

B. Hemochromatosis

1. General

- a. Increase total body iron
- b. Deposition in heart, liver, pituitary, pancreas, gonads, skin
- c. Iron deposits **within** myocardial cells (storage disease)
- d. 1° (autosomal recessive); inappropriate increased absorption from GI tract
- e. 2° (transfusion, Fe-therapy, thalassemia major, hemolytic anemias, alcoholic liver disease, etc)

2. Echo findings

- a. Ventricular wall thickness usually normal
- b. Diastolic dysfunction in early stage
- c. Dilated cardiomyopathy common
- d. Restrictive cardiomyopathy uncommon
- e. Mild valvular regurgitation

C. Sarcoid

1. General

- a. Multisystem granulomatous disease of unknown cause
- b. Lungs, skin, heart, reticuloendothelial system
- c. Young adults (75% <40 yrs old)

2. Cardiac manifestations

- a. Sudden cardiac death
- b. Arrhythmias
- c. Conduction abnormalities
- d. LV dysfunction and CHF
- e. Cor pulmonale (2° pulmonary sarcoid)

3. Echo features

- a. Dilated cardiomyopathy (4-chamber enlargement)
- b. Regional WMA
 - 1) Focal septal thinning ± aneurysm
 - 2) Basal posterolateral wall thinning ± aneurysm

- c. Diastolic dysfunction precedes systolic
- d. If cor pulmonale
 - 1) Pulmonary hypertension
 - 2) RV and RA enlargement
- e. Pap muscle dysfunction and MR
- f. Pericardial effusion uncommon (pericarditis)
- g. RCM if myocardial infiltration

D. Amyloid

A. General

- 1. Heterogeneous group of diseases
- 2. Deposition of extracellular proteins (amyloid) in various organs
 - a. Unique beta-pleated sheet conformation
 - b. Amyloid deposits in interstitium **between** myocardial cells
- 3. Types of amyloid
 - a. Primary (fibrils derived from immunoglobulin light chain)
 - b. Secondary
 - 1) Associated with chronic diseases (eg RA, Tbc)
 - 2) Not immunoglobulin light chain
 - c. Familial
 - d. "Senile"
 - 1) 10% of autopsies pts > 75 years
 - 2) Cardiac involvement may be extensive
- 4. Cardiac involvement common
 - a. 50% of 1° systemic amyloidosis

5. Clinical manifestations

- a. Can be asymptomatic
- b. CHF: diastolic and/or systolic dysfunction
- c. Rhythm abnl (AF)
- d. Conduction disturbances
- e. Embolic events (atrial, LV, AF)
- f. Coronary insufficiency (amyloid infiltration of intramural coronary arteries)

6. Echo features

- a. Increased LV/RV wall thickness
- b. Increased myocardial echogenicity (“granular sparkling”)
- c. RCM S
 - 1) Systolic fx preserved early, poor late
 - 2) Diastolic dysfunction
 - a) $E/A > 2$
 - b) Rapid decel time
- d. Valvular thickening and regurg (usually mild)
- e. Atrial thrombus
- f. Pericardial effusion

E. Connective tissue diseases

1. Rheumatoid arthritis

a. General

- 1) Most common connective tissue disease
- 2) Age-related (ages 20-60 years)
- 3) Females > males (2 to 4:1)
- 4) Cardiac abnormalities in 30-50% (necropsy)
- 5) Cardiac disease often subclinical during life

b. Cardiac

- 1) Pericardial
- 2) Myocardial
- 3) Endocardial/valvular
- 4) Conduction system

5) Aortic and pulmonary

b. Echo features

1) Pericardial (\approx 50% of RA patients)

- a) Effusion (acute pericarditis)
- b) Constrictive pericarditis (<10%)
- c) Effusion-constrictive

2) Myocardial

- a) Global LV dysfunction (myocarditis)
- b) Regional WMAs, rare (MI from coronary arteritis)
- c) Diastolic dysfunction

- 1) Impaired relaxation
- 2) Not uncommon

- d) Secondary amyloidosis
- e) Nodules in myocardium

3) Valvular/endocardial

- a) Valve thickening & regurg (valvulitis)
- b) Nodules
- c) AR 2° aortic enlargement

- 4) Aortic aneurysm, wall thickening: 2° aortitis (rare)
- 5) Secondary pulmonary hypertension (rare)

2. Systemic lupus erythematosus

a. General

- 1) Common autoimmune disease (1/2000)
- 2) More prevalent /severe women (5-10:1), blacks (3:1)
- 3) Cardiac involvement >40%
- 4) Most common causes death: heart disease, infections, renal disease

b. Cardiovascular

- 1) Valvular involvement common (20-75%)
- 2) Pericardial involvement common
 - a) Pericarditis > 50%
 - b) Effusion 10-15%
 - c) Tamponade < 1%
- 3) Myocardial
- 4) Pulmonary hypertension (up to 5%)
- 5) Aortitis (rare)
- 6) Coronary artery aneurysms (rare)
- 7) Vascular thrombosis

c. Valvular – Echo features

- 1) Thickening (esp mitral, aortic)
- 2) Nodularity
- 3) Regurgitation
- 4) Non-bacterial vegetations (Libman-Sacks)
 - a) Usually <1 cm²
 - b) Irregular borders
 - c) No independent motion
- 5) MV prolapse (5-10%)

d. Pericardial – Echo features

- 1) Effusion (often clinically silent)
- 2) Cardiac tamponade uncommon

e. Myocardial – Echo features

- 1) Global LV dysfunction
- 2) Regional WMA
 - a) Accelerated atherosclerosis
 - b) Coronary vasculitis
 - c) Coronary embolism

3. Antiphospholipid Syndrome

a. General

- 1) High titres of antiphospholipid antibodies
- 2) 1° or 2°
- 3) Clinical features
 - a) Recurrent arterial & venous thromboembolism
 - b) Thrombocytopenia
 - c) Spontaneous abortion

b. Echo features

- 1) Intracardiac, aortic thrombi
- 2) LV systolic dysfunction
 - a) RWMA 2° MI
 - b) Dilated cardiomyopathy
- 3) Valvular regurgitations
- 4) Pulmonary hypertension

4. Progressive systemic sclerosis (scleroderma)

a. General

- 1) Excess connective tissue accumulates in blood vessels, skin, joints, skeletal muscle, heart
- 2) Two major clinical varieties
 - a) Diffuse (20%)
 - b) Limited cutaneous (80%)
- 3) Women >men (3:1); ages 30-50 years

b. Echo features

- 1) Pulmonary hypertension
- 2) Pericardial
 - a) Effusion, tamponade, constriction
 - b) CREST – symptomatic pericarditis (30%)

3) Myocardial

- a) LVH with systolic hypertension
- b) LV dysfunction (up to 75%)
- c) Cardiomyopathy (DCM or RCM)

5. Mixed connective tissue disease (MCTD)

- a) Features of SLE, RA, scleroderma, polymyositis
- b) Cardiac features
 - 1) Pericarditis
 - 2) Coronary arteritis (rare)
 - 3) Myocarditis (rare)
 - 4) Pulmonary hypertension 2° pulmonary disease
 - 5) Mitral valve prolapse

F. Ankylosing spondylitis

1. General

- a. An HLA-B27-related autoimmune disease
- b. Inflammation of vertebral and sacroiliac joints, peripheral arthritis, anterior uveitis
- c. M > F (3:1)
- d. Aortic disease common (up to 10%)
- e. Conduction abnormalities common (up to 33%)
- f. WPW

2. Echo features

- a. Dilatation of aortic annulus and sinus of Valsalva
- b. Aortic valve thickening
- c. Aortic regurgitation
- d. Thickening of aorto-mitral junction (“subaortic bump”)
- e. Mitral valve prolapse
- f. LV systolic dysfunction
- g. Pericarditis/pericardial effusion (rare)

G. Reiter’s syndrome

1. General

- a. Similar echo findings to ankylosing spondylitis
- b. Pericarditis more common
- c. Conduction abnormalities (up to 25%)

2. Echo findings (see ankylosing spondylitis)

H. Psoriatic arthritis

1. Mitral valve prolapse
2. Aortic disease less common than ankylosing spondylitis

I. Marfan's syndrome

1. General

- a. 70% hereditary
 - 1) Autosomal dominant with high penetrance
 - 2) 30% new mutation
- b. Involves ocular, skeletal, and C-V systems
- c. Most common cause of death in adults is aortic dissection

2. Echo findings

- a. Aortic root dilatation
 - 1) Dilated aortic annulus
 - 2) Dilated sinuses of Valsalva
- b. Dilated ascending aorta
- c. Fusiform ascending aortic aneurysm (annuloaortic ectasia)
- d. Aortic regurgitation
- e. Aortic dissection
- f. Myxomatous mitral valve and MVP
- g. Mitral regurgitation
- h. Dilated and calcified mitral annulus

J. Giant cell arteritis

1. General

- a. Vasculitis involving large and medium-sized arteries
- b. Age >50 years
- c. Increased risk of developing aortic aneurysm

2. Echo findings

- a. Aortic aneurysm and dissection
- b. Dilatation and thickening of aortic valve and cusps
- c. LV systolic dysfunction from myocarditis
- d. Pericardial effusion (pericarditis)

K. Takayasu arteritis

1. General

- a. Granulomatous panarteritis of large vessels
- b. Unknown etiology
- c. Typically women <40 years old

2. Echo findings

- a. Dilatation of aorta
- b. Aortic regurgitation
- c. Stenosis and occlusion of large vessels

L. Kawasaki's disease

1. General

- a. Acute systemic vasculitis of unknown origin
- b. Mucocutaneous, lymph node syndrome
- c. Usually <5 years of age (80%)
- d. Cardio

2. Cardiovascular

- a. Vasculitis of coronary vasa vasorum
- b. Leads to coronary artery aneurysms
 - 1) Thrombosis
 - 2) Stenosis
 - 3) Myocardial ischemia, myocardial infarction
- c. Conduction abnormalities

3. Echo findings

a. Coronary artery aneurysms (15-25%)

- 1) Small <4 mm
- 2) Medium 4-8 mm
- 3) Giant >8 mm

b. Pericardial effusion (pericarditis) –30%

c. Myocarditis (common)

d. Mitral regurgitation

M. Syphilitic aortitis

1. General

- a.
- b. Aortitis ± coronary ostial stenosis
- c. Coronary artery aneurysms (see Section 6, II, B, 4)

2. Echo findings

- a. Dilated aortic root: aneurysm
- b. Aortic regurgitation
- c. Aortic dissection
- d. Aorto-pulmonary fistula

N. Hypereosinophilic syndrome (Loeffler's)

1. General

- a. Multisystem disease: cardiac, skin, neurologic, eyes, pulmonary, GI, hepatic, renal, musculoskeletal
- b. Non-tropical form of endomyocardial fibrosis
- c. Always associated with hypereosinophilia

2. Echo findings

- a. LV >RV apical cavity obliteration
- b. Ventricular thrombus
- c. Cardiomyopathy
 - 1) Restrictive
 - a) Biatrial enlargement
 - b) Normal LV and RV size and systolic function
 - c) Restrictive hemodynamics
 - 2) Dilated cardiomyopathy (diffuse myocarditis)
 - d. Thickening/obliteration of inferobasal mitral inflow tract (“entraps”, “plasters down” posterior leaflet)
 - e. Mitral regurgitation (often mod-to-severe)
 - f. Variable severity of TR
 - g. Pericardial effusion (pericarditis)
 - h. Uncommon
 - 1) Constrictive pericarditis
 - 2) Asymmetric septal hypertrophy

O. Churg – Strauss syndrome

1. General

- a. Systemic vasculitis
- b. Peripheral and extravascular eosinophilic
- c. Associated asthma and/or allergic rhinitis

2. Cardiac/echo findings

- a. Pericardial effusion (pericarditis)
- b. Dilated cardiomyopathy
- c. Endomyocardial fibrosis

P. Wegener's Granulomatosis

1. General

- a. Disease of unknown etiology
- b. Protean clinical manifestations
- c. Multisystem involvement
- d. Cardiac involvement:
 - 1) Pericarditis
 - 2) Myocarditis
 - 3) Valvulitis
 - 4) Arteritis
 - 5) Mass lesions (granulomas)
 - 6) Arrhythmias

2. Echo findings

- a) LV regional WMA
- b) Global LV hypokinesis
- c) Pericardial effusion
- d) Valvular regurgitation
- e) Left atrial mass (uncommon)

Q. Whipple's disease

R. Endocrine diseases: Cardiovascular and echo findings

1. Hyperthyroidism

- a. Increased SV, CO, and LV mass
- b. Dilated CM (tachycardia-induced)
- c. Diastolic dysfunction (impaired relaxation)
- d. Atrial fibrillation
- e. Pulmonary hypertension (rare)

2. Hypothyroidism

- a. Decreased heart rate and CO
- b. Prolonged diastolic relaxation
- c. Dilated cardiomyopathy
- d. Pericardial effusion (tamponade rare)
- e. Valvular thickening
- f. Accelerated atherosclerosis

3. Pheochromocytoma

- a. LV systolic dysfunction
(catecholamine-induced)
- b. LV hypertrophy
- c. Reversible dilatation
- d. HCM with/without dynamic LVOT obstruction

4. Acromegaly

S. HIV disease (AIDS)

1. General

2. Echo findings

a. Pericardial

1) Pericardial effusion \pm tamponade (up to 40%)

- a) Infectious (Tbc, bacterial, fungal, viral)
- b) Malignant (lymphoma, Kaposi's, metastatic)
- c) Non-HIV related cause

2) Pericardial constriction

b. Dilated cardiomyopathy (up to 50%)

- 1) Myocarditis (HIV, bacterial, fungal, Tbc)
- 2) Neoplastic infiltration (lymphoma, Kaposi's)
- 3) Alcohol, nutritional deficiencies

c. Cardiac masses

- 1) Neoplasms (Kaposi's, lymphoma)
- 2) Vegetations

- a) Marantic endocarditis (common)
- b) Infective endocarditis

d. Pulmonary hypertension

- 1) Recurrent pulmonary infections
- 2) HIV-related interstitial pneumonitis and fibrosis
- 3) Necrotizing angiitis 2° drug use
- 4) Thromboembolic events

T. Ergot alkaloids, appetite suppressants

U. Systemic infection/sepsis

1. General

2. Echo findings

- a. Reversible dilated LV with systolic dysfunction (myocardial depression)
- b. Diastolic dysfunction
- c. Vegetation
- d. Pericardial and pleural effusions

V. Hematologic disorders

W.

X. Miscellaneous conditions

1. Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu)

a. General

- 1) Triad: mucocutaneous and visceral telangiectasias, recurrent epistaxis, and familial history
- 2) Autosomal dominant

b. Cardiac involvement

- 1) High cardiac output
- 2) Pulmonary A-V malformations (hypoxemia)
- 3) Coronary A-V malformations (rare)

c. Echo findings

- 1) Contrast echo → delayed contrast in left atrium

III. Trauma

A. Blunt trauma

B. Penetrating trauma

Sources Used in Compiling Core Curriculum

A. Textbooks

1. Nanda NC ed. Doppler Echocardiography. 2nd edition, Lea and Febiger, Philadelphia, 1993.
2. Roelandt JRTC, Sutherland GR, Iliceto S, Linder DT, eds. Cardiac Ultrasound. Livingston, 1993.
3. Feigenbaum H Echocardiography. 5th edition, Lea and Febiger, Philadelphia, 1994.
4. Weyman AE. Principles and Practice of Echocardiography. Lea and Febiger, Philadelphia, 1994.
5. Otto CM, Pearlman AS. Textbook of Clinical Echocardiography W.B. Saunders Company, Philadelphia, 1995.
6. Marwick TH. Cardiac Stress Testing and Imaging. Churchill Livingstone, New York, 1996.
7. Harry MJ. Cardiac Doppler Hemodynamic Workbook, 1997.
8. Otto CM. The Practice of Clinical Echocardiography. W.B. Saunders Company, Philadelphia, 1997.

B. Guidelines

1. American Society of Echocardiography Committee on Standards, Subcommittee on Quantification of Two-Dimensional Echocardiograms. Recommendations for the left ventricle by two-dimensional echocardiography. J Am Soc Echocardiography 1989;2:358-367.
2. ACC/AHA Task Force on Practice guidelines, Committee on Clinical Applications of Echocardiography: ACC/AHA guidelines for the clinical applications of echocardiography. Circulation 1997;95:1686-1794.
3. Gibbons RJ, Balady GJ, Beasley JW, et al. ACC/AHA guidelines for exercise testing. A report of the ACC/AHA Task Force on Practice guidelines. J Am Coll Cardiol 1997;30:260-315.
4. ACC/AHA Guidelines for the Management of Patients with Valvular Heart Disease. A report of the ACC/AHA Task Force on Practice Guidelines (Committee of Management of Patients with Valvular Heart Disease). Circulation 1998;32:1456-1588.

5. ASE/SCA Guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination; Recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. J Am Echocardiogr 1999;12:884-900

C. Miscellaneous sources

1. ACC Core Cardiology Training Symposium (COCATS): Guidelines for training in adult cardiovascular medicine. J Am Coll Cardiol 1995;27:1-334.
2. ARDMS Exam content outline.
3. Content outline: perioperative transesophageal echo. Society of Cardiovascular Anesthesiologists.
4. Handouts from various speakers at various Echo Symposia and Conferences.

