APCDE 2011

Valvular Heart Disease (2)

# LV geometric and functional changes in VHD: How to assess?

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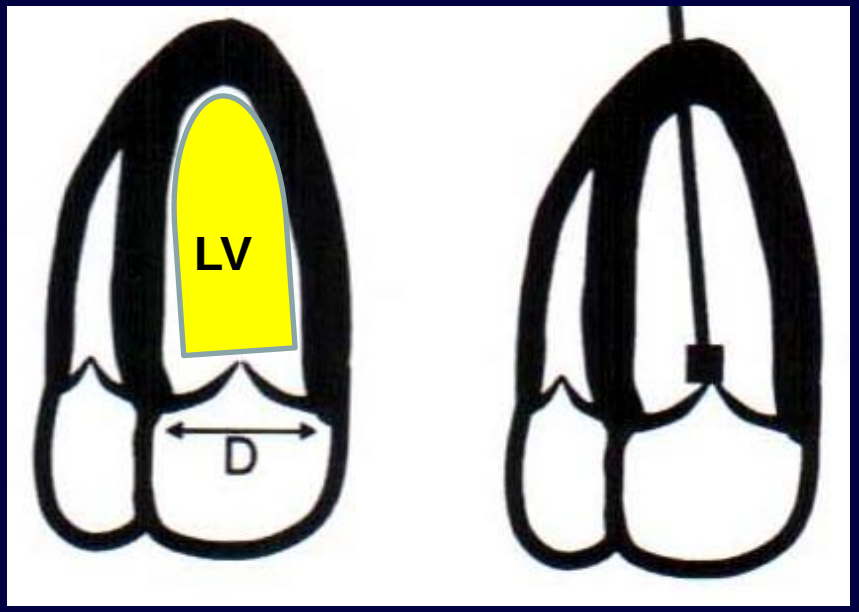
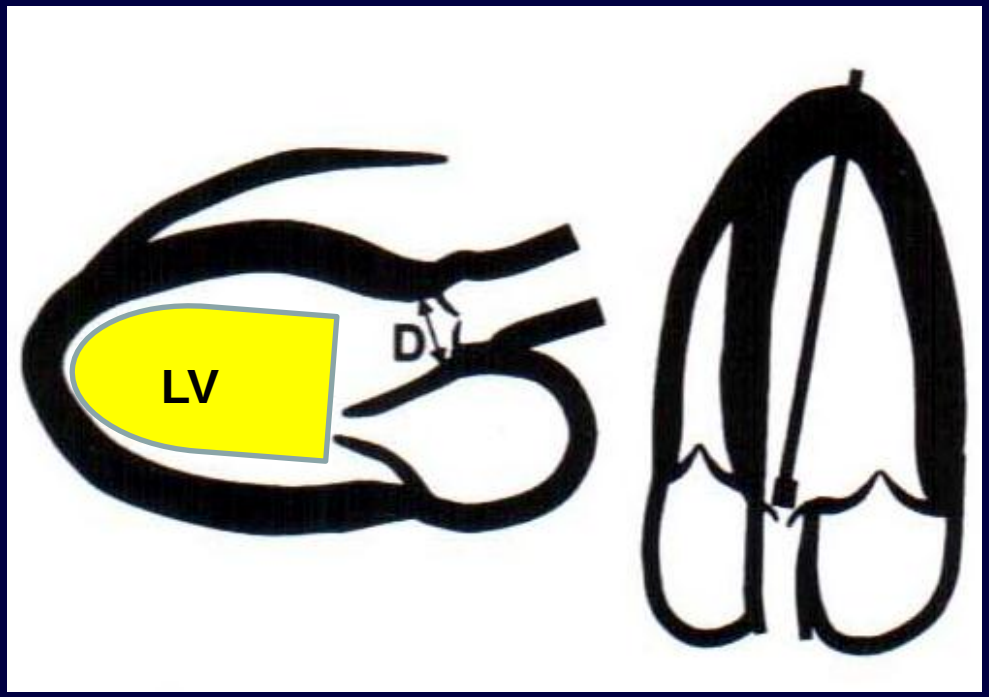



LV inflow across MV




LV outflow across AV

# LV geometric changes

## Pressure overload

Increase systolic stress (Afterload)

⇒ Parallel addition of new sarcomeres

⇒ Wall thickening

⇒ Concentric Hypertrophy

## Volume overload

Increase diastolic stress (Preload)

⇒ Series addition of new sarcomeres

⇒ Chamber enlargement

⇒ Eccentric Hypertrophy

# LV geometric changes

## Pressure overload

Increase systolic stress (Afterload)

⇒ Parallel addition of new sarcomeres

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Increase diastolic stress (Preload)

⇒ Serial addition of new sarcomeres

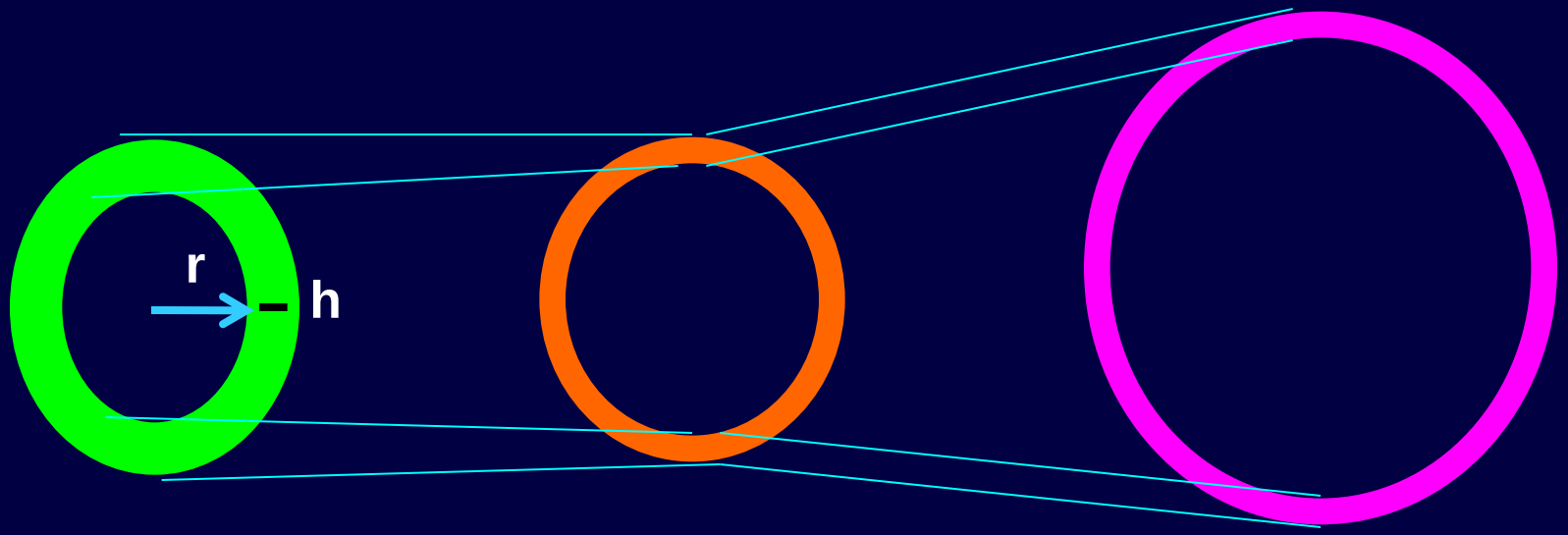
⇒ Chamber enlargement

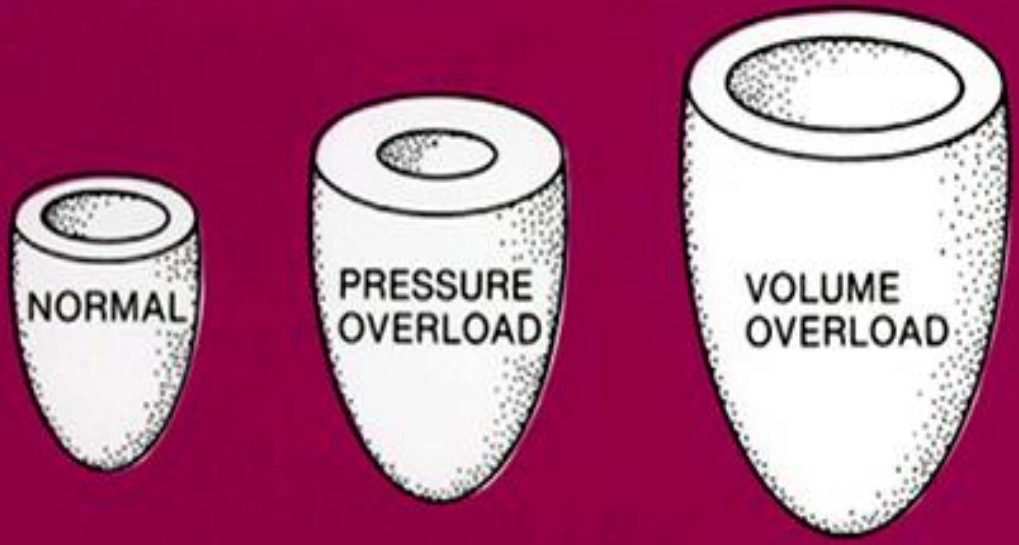
⇒ Eccentric Hypertrophy

**Concentric** **Normal** **Eccentric**

Wall stress = Force/Area = Pressure x r/h ; Laplace's Law

| | Concentric | Eccentric |
|---|---|---|
| r | ↓ | ↑ |
| h | ↑ | ↑ → |
| δ | ↓ | ↑ |
| EF | ↑ → | ↓ |

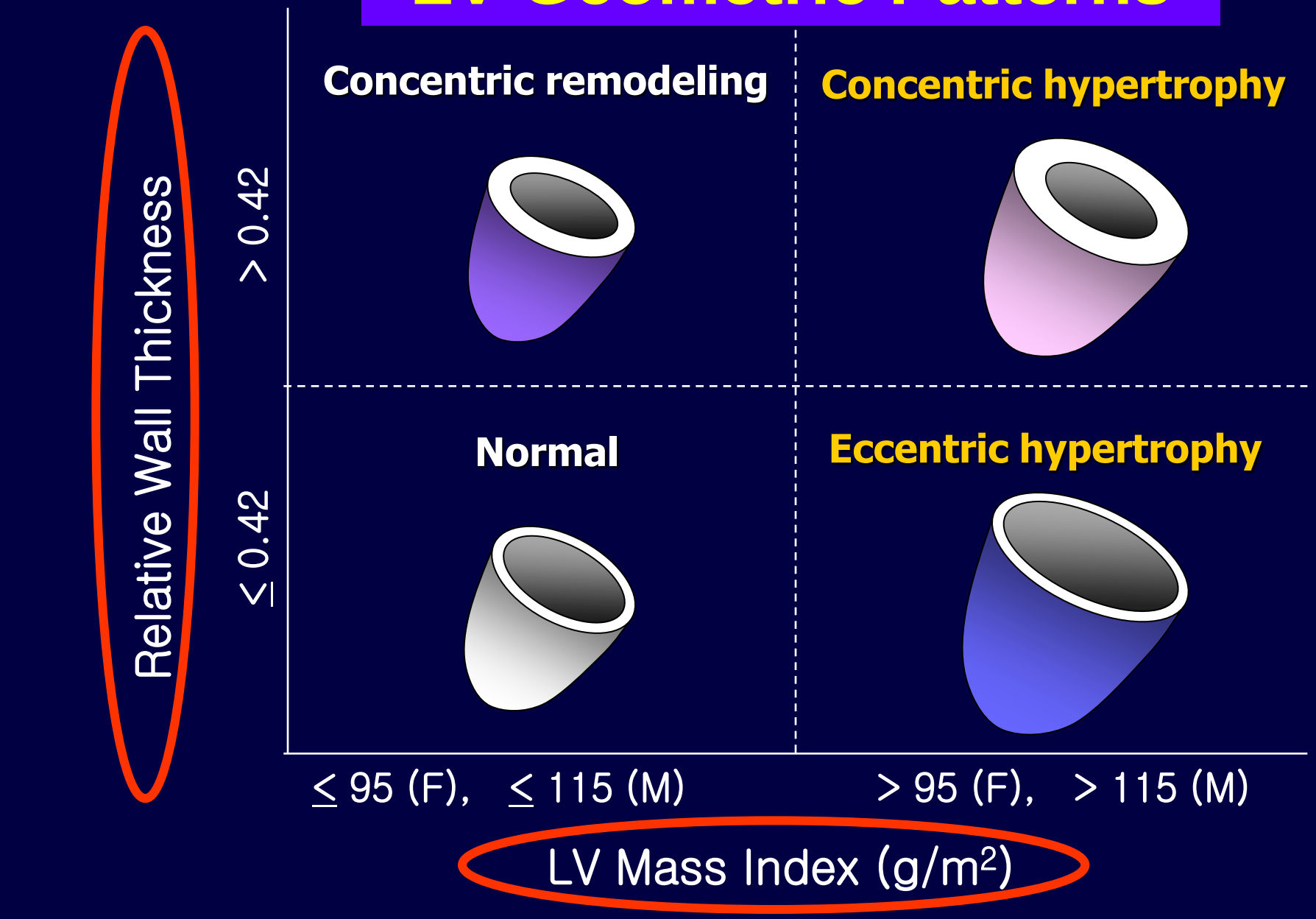





| | | | |
|---|---|---|---|
| LV Pressure (mm Hg) | 117 ± 7/10 ± 1 | 226 ± 6*/23 ± 3* | 138 ± 7/23 ± 2* |
| LVMI ($gm/m^2$) | 71 ± 8 | 206 ± 17* | 196 ± 17* |
| LV wall thickness (mm) | 8.2 ± .6 | 15.2 ± .9* | 10.6 ± 5* |
| $\sigma_m$ ($10^3$ dynes/$cm^2$) | | | |
| Peak systolic | 151 ± 4 | 161 ± 24 | 175 ± 7 |
| End diastolic | 17 ± 2 | 23 ± 3 | 41 ± 3* |

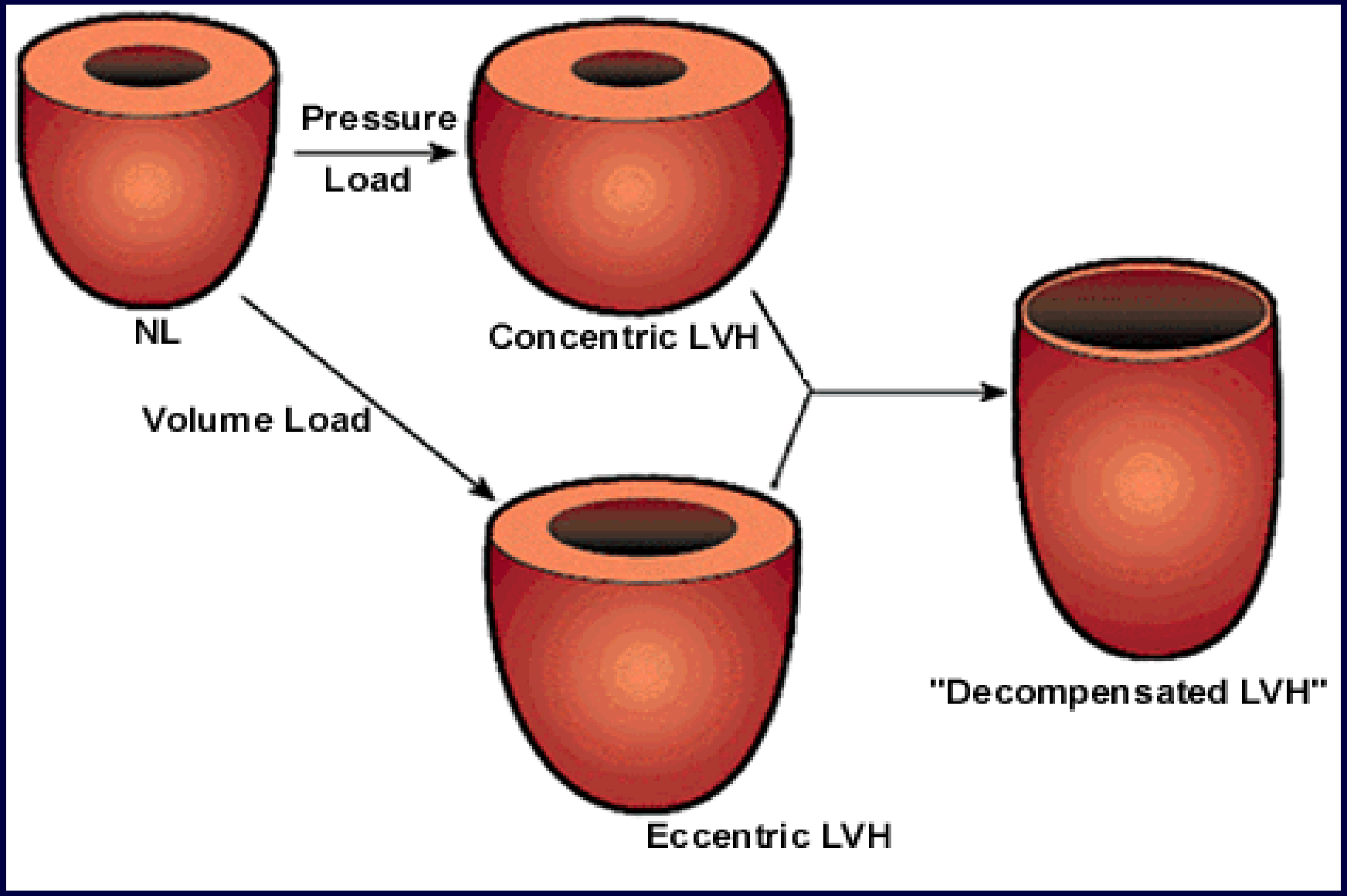
*$p < .01$

# LV Geometric Patterns




Relative wall thickness (RWT) = (2PWTd)/LVIDd

# Schematic representation of LVH



# Preload and Afterload in VHD

- **Increased Preload**

  **MR, AR**
- **Decreased Preload**

  **MS**
- **Increased Afterload**

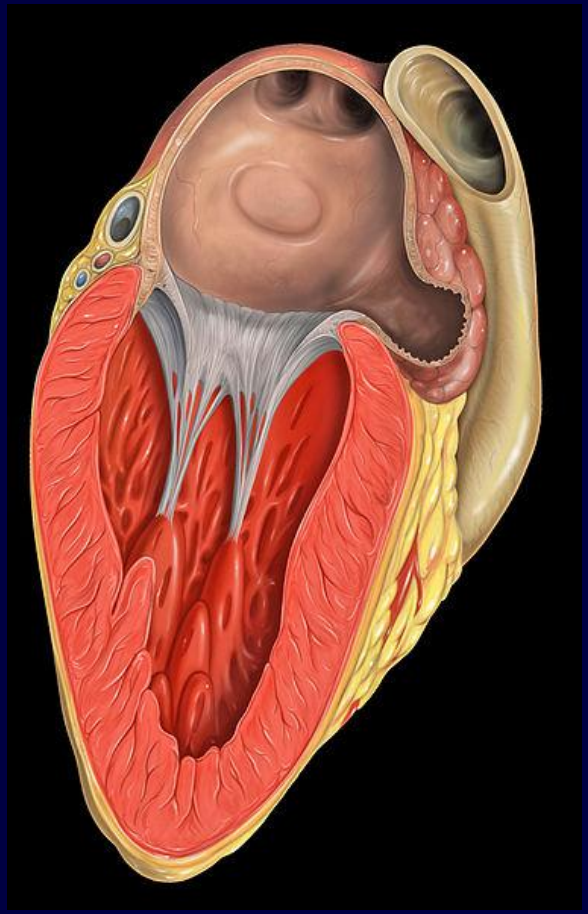
  **AS**
- **Decreased Afterload**

  **MR**

# LV changes in VHD

**Chronicity**

**Severity**



LV geometric changes

LV functional changes

How to assess?

# LV geometric change

Dimension (M-mode or 2D-Guided)

Volumes

- Biplane Simpson's
- Area length

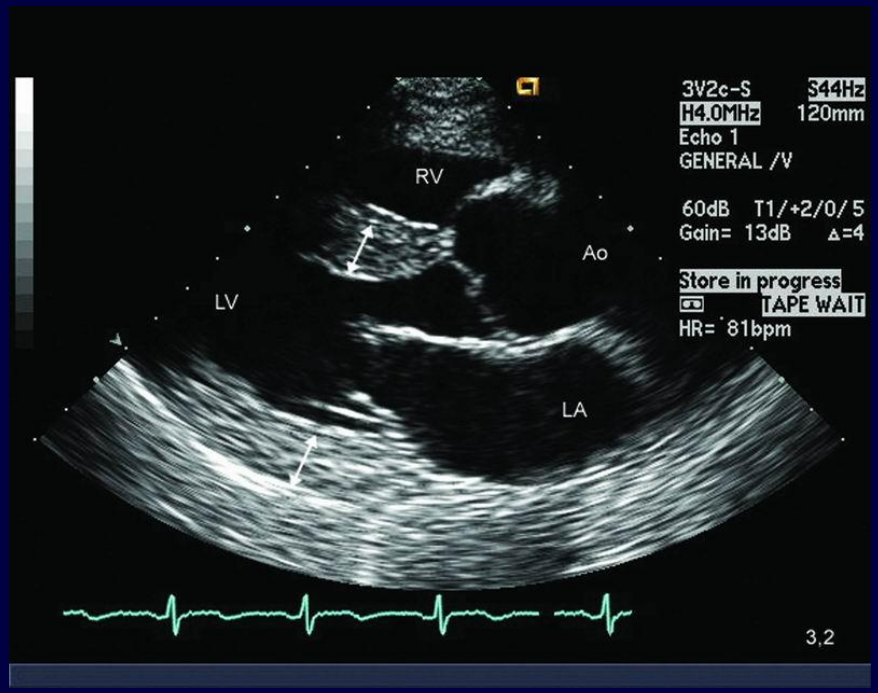
Mass

- M-mode or 2D-guided
- Area length
- Truncated ellipsoid

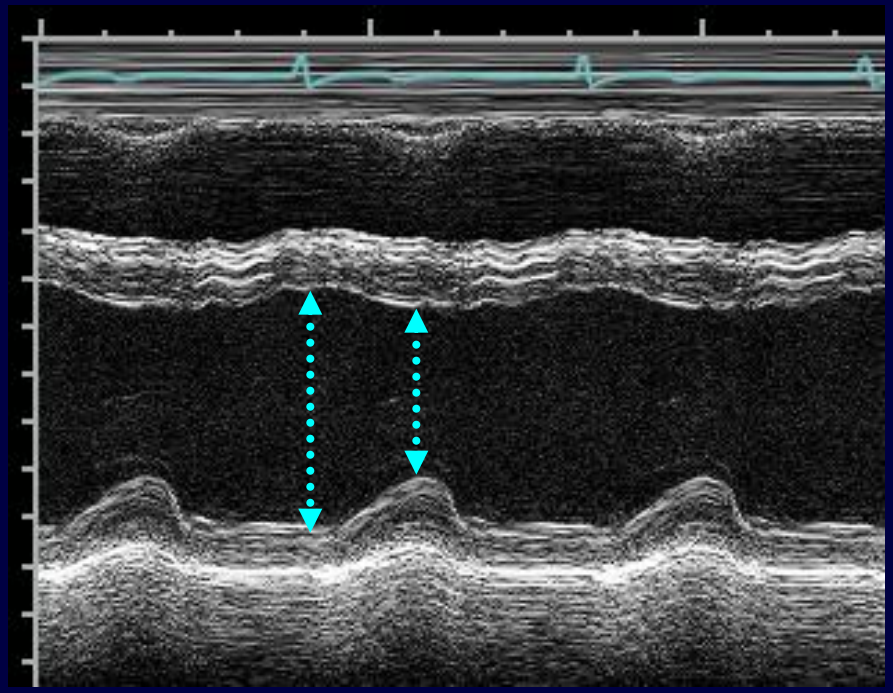
Sphericity index

# LV mass

## 2D




## M-mode



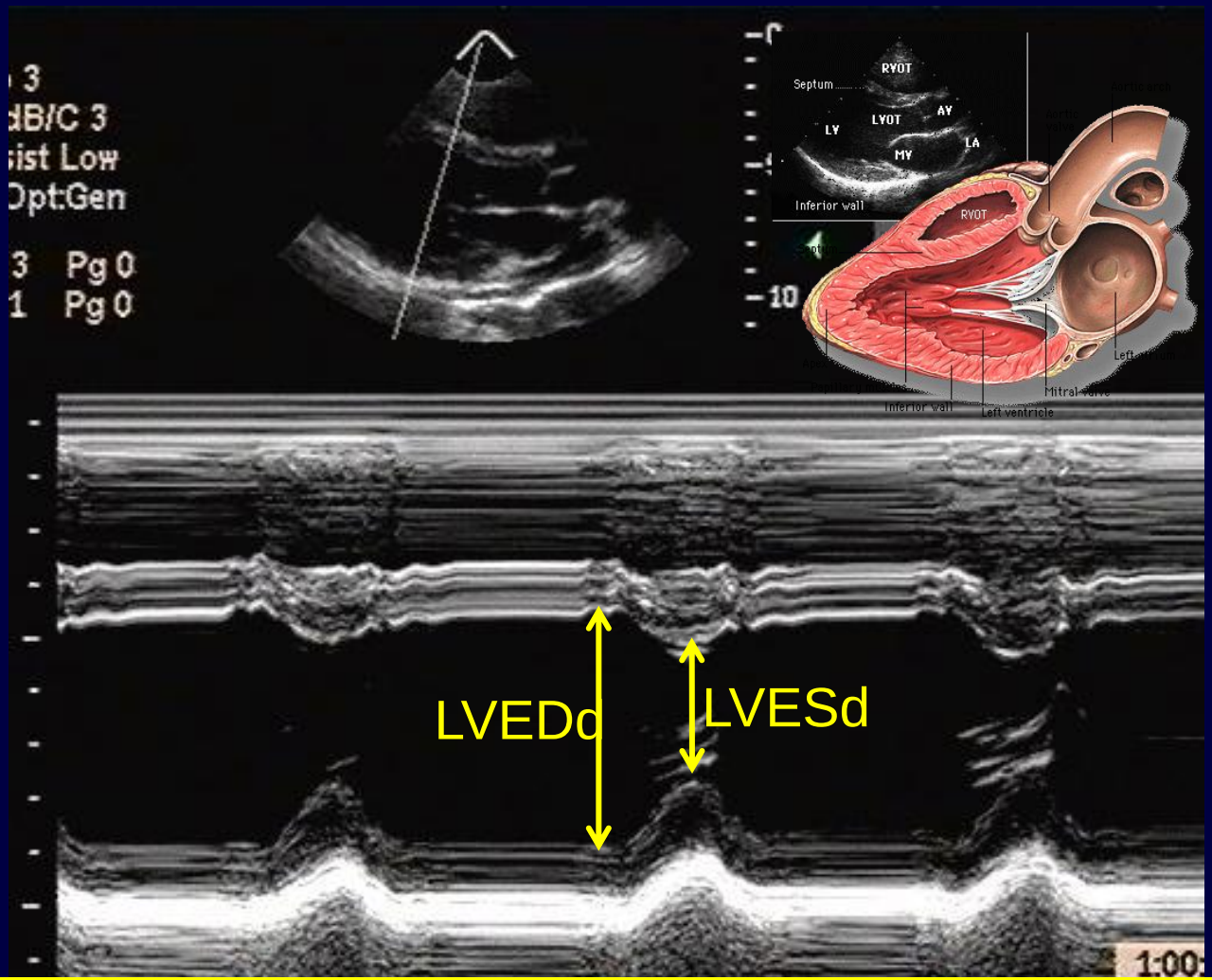
LV mass (g)= $1.04x[(LVID+PWT+IVST)^3 - LVID^3)]x0.8+0.6$

Devereux RB, et al. ***Am J Cardiol 1986;57:450***

# LV systolic function

- Fractional shortening (FS)
- Ejection fraction (EF)
- Stroke volume / cardiac output
- dP/dt
- Mitral annular systolic wave (Sm) by TDI
- Mitral E point septal separation (EPSS)
- Tei index (Index of myocardial performance, IMP

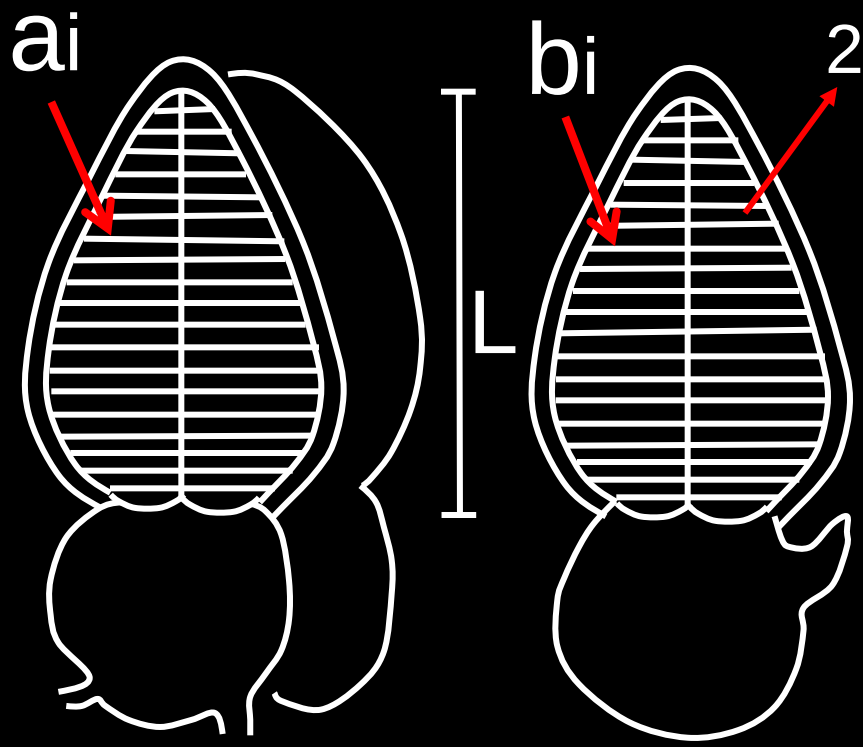
# FS and EF




$FS = [(LVEDd - LVESd) / LVEDd] \times 100\ (\%)$

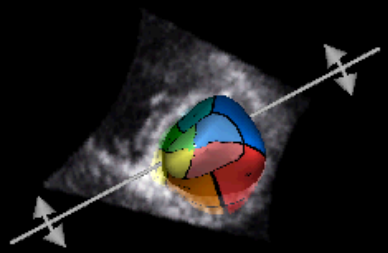
$EF = [(LVEDd^2 - LVESd^2) / LVEDd^2] \times 100\ (\%)$

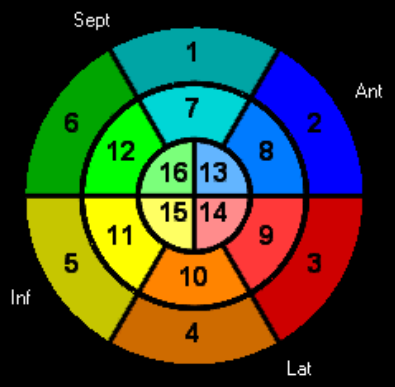
# EF: modified Simpson method & 3D

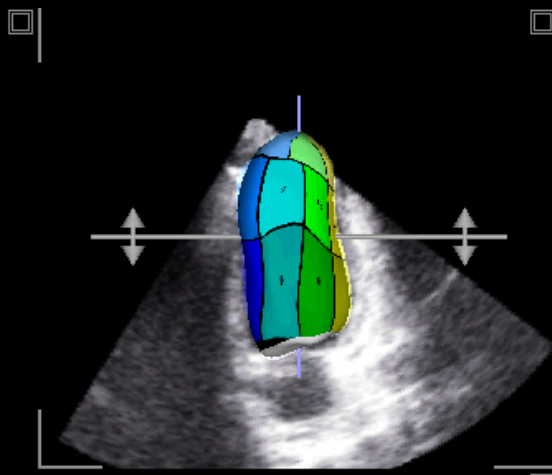



$$V = \pi/4 \sum_{i=1}^{20} aibi \cdot L/20$$

Summation of 20 discs
= LV volume





| | |
|---|---|
| EDV | 150.32 ml |
| ESV | 67.50 ml |
| SV | 82.83 ml |
| EF | 55.10 % |
| SDI16 | 45 ms |

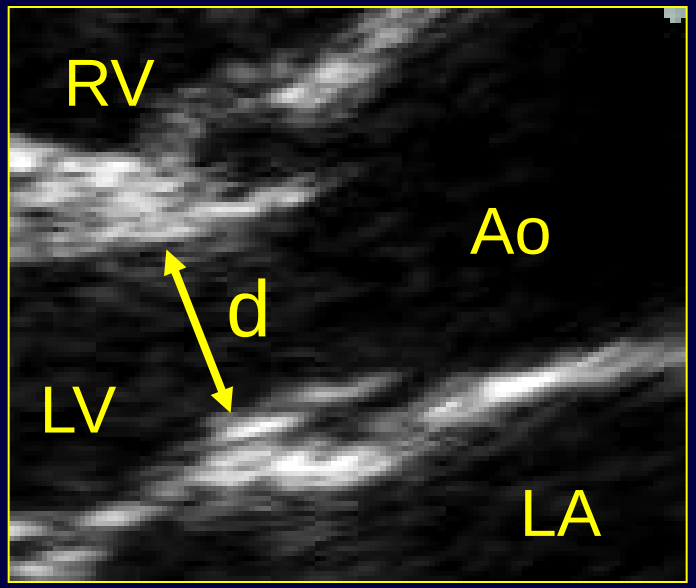
# LV EF

- Do not reflect diastolic function
- Overestimated in the case of MR
- Not relevant to the prognosis
- Not relevant to exercise capacity

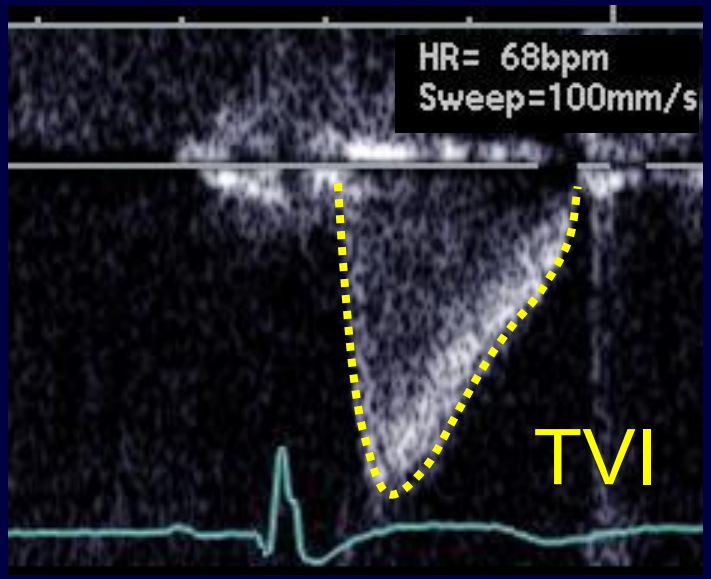
# Stroke Volume

SV = LVOT area X LVOT TVI

**SV =**

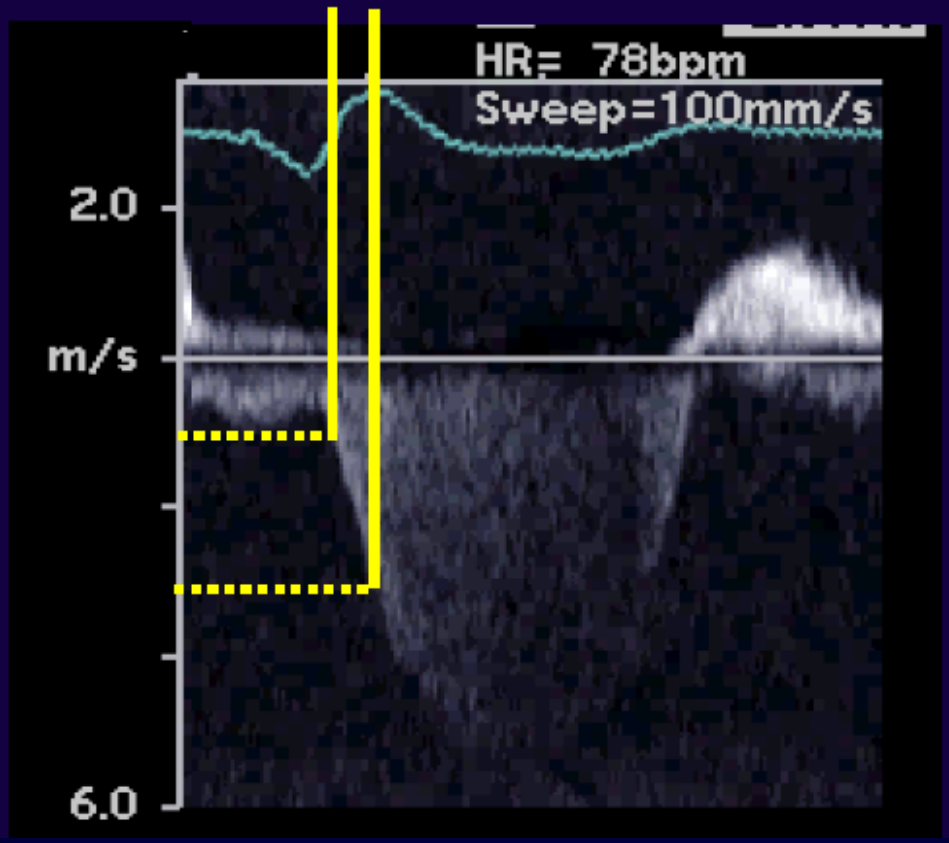



**X**



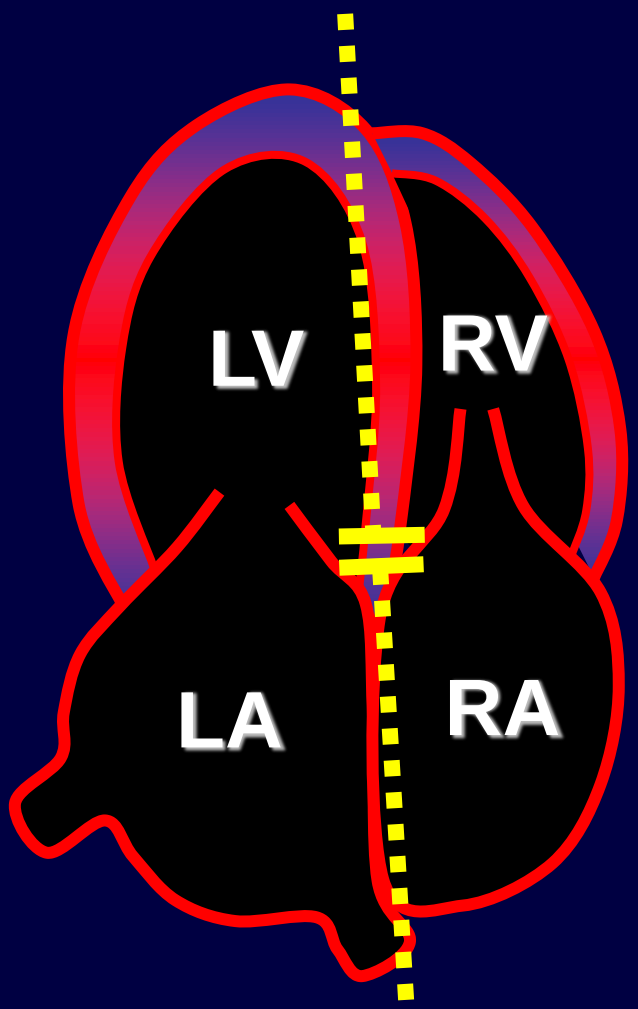
# Assessment of LV function

## *(+) dP/dt*

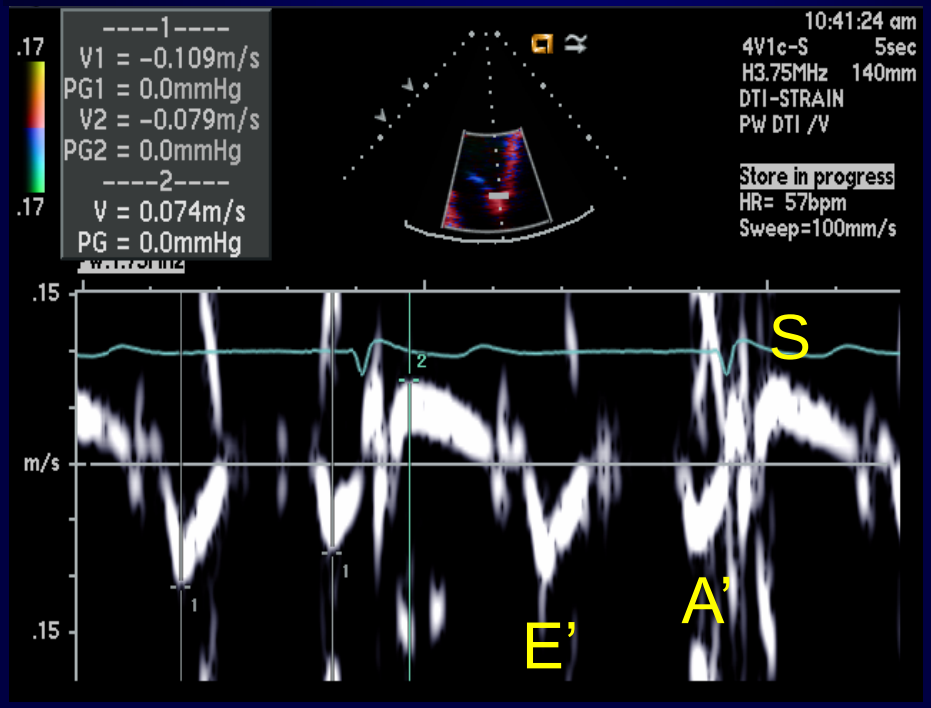



- 1m/s = 4 mmHg
- 3m/s = 36 mmHg
- $\Delta$1-3 = 32 mmHg
- dP/dt = 32/t (s)
  = 32/0.050
  = 640 mmHg/s

# Mitral annular velocity




- Systolic (S) velocity
- Early diastolic (Ea, Em, E', e') velocity
- Late diastolic (Aa, Am, A', a') velocity
- E/e' ratio

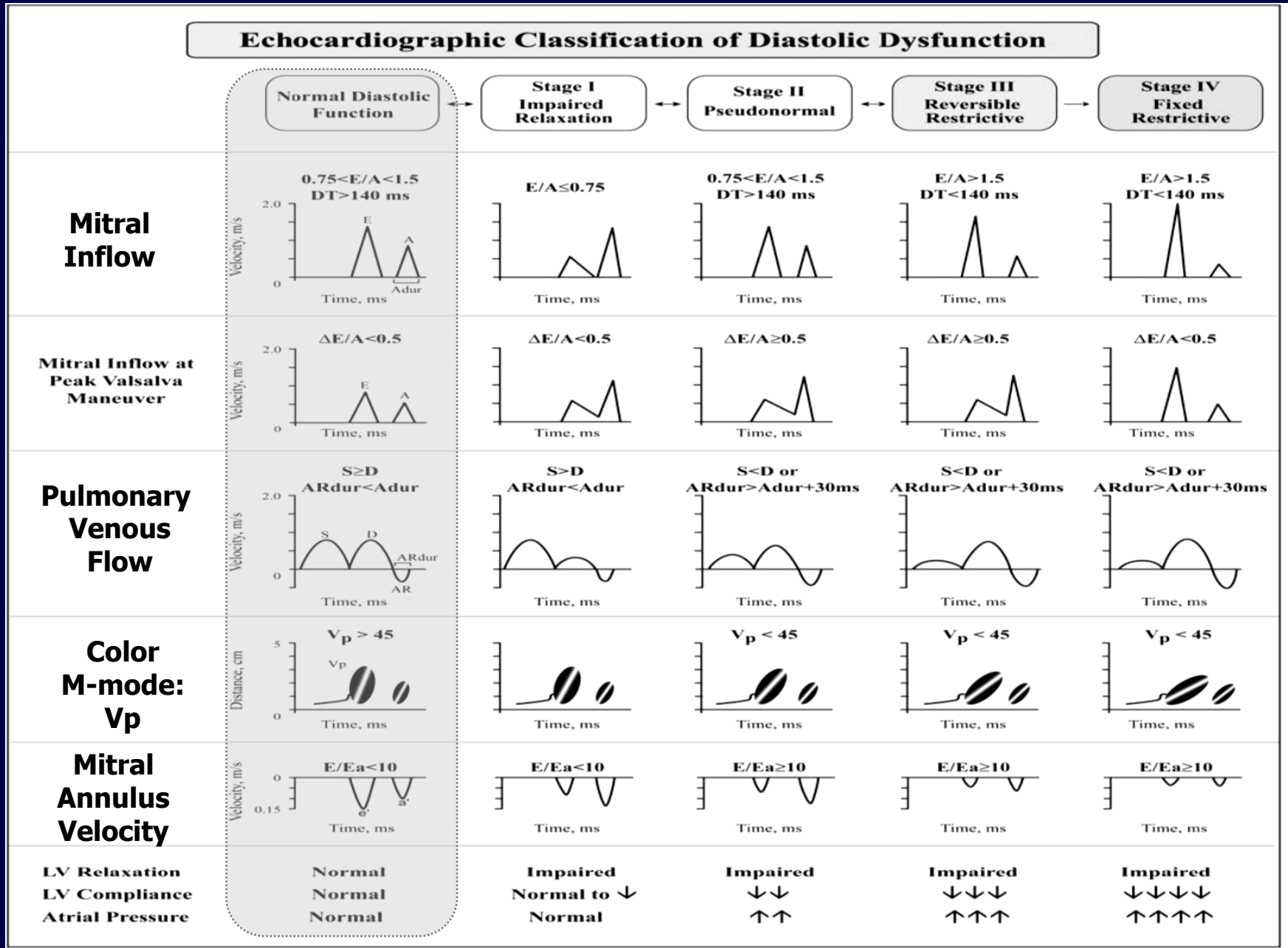



- **Close correlation between S and LVEF**
- **S > 7.5 cm/s: Predict LVEF > 50% (sensitivity: 79%, specificity: 88%)**

# Determinants of LV diastolic function

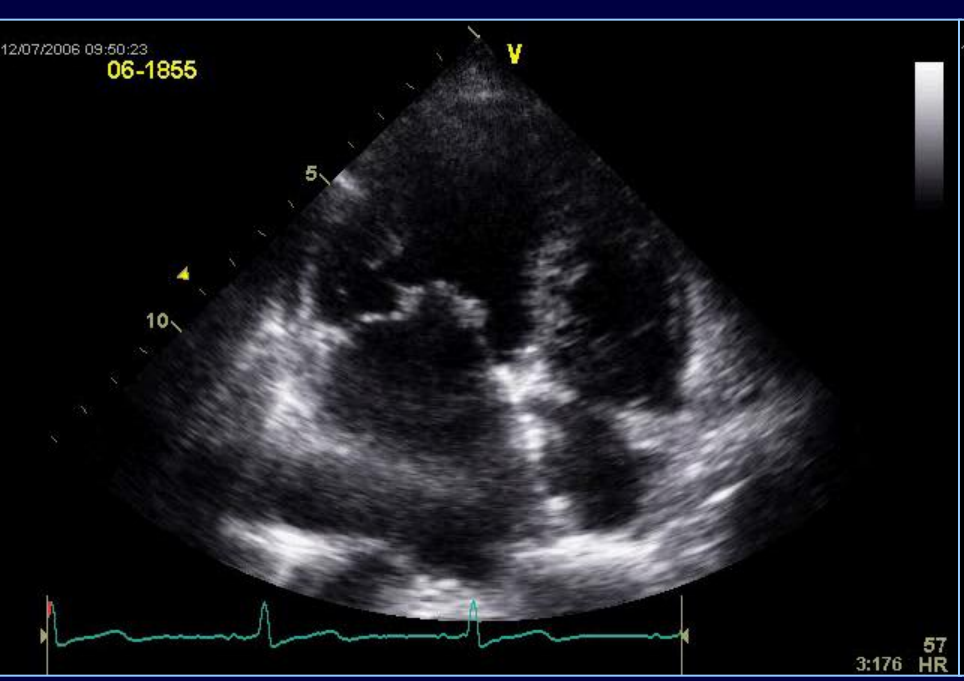
- LV elastic recoil
- LV active relaxation
- Atrial contraction

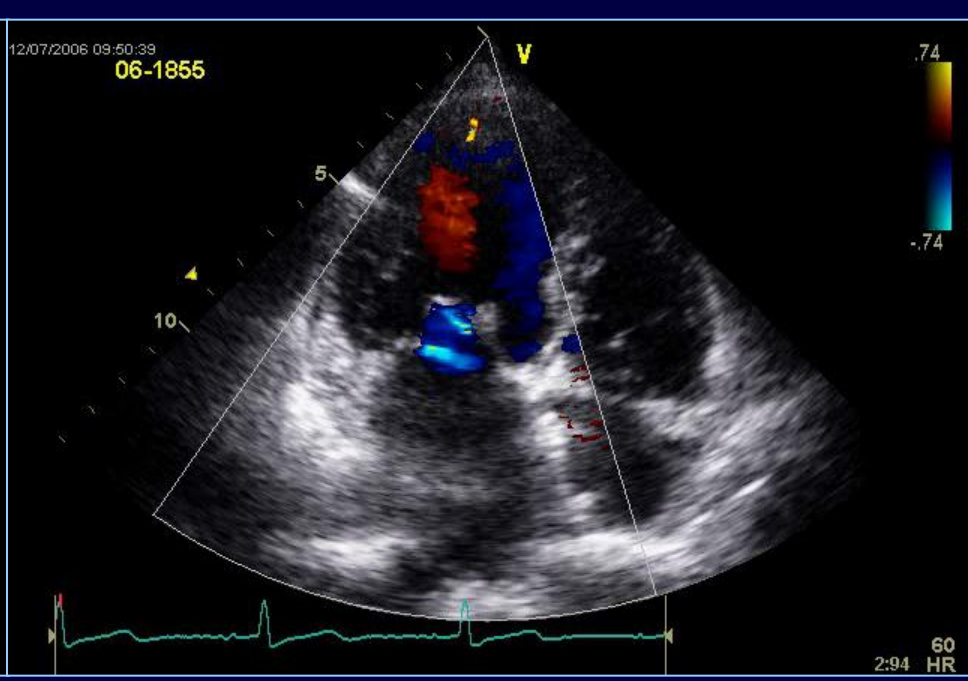
# LV diastolic function

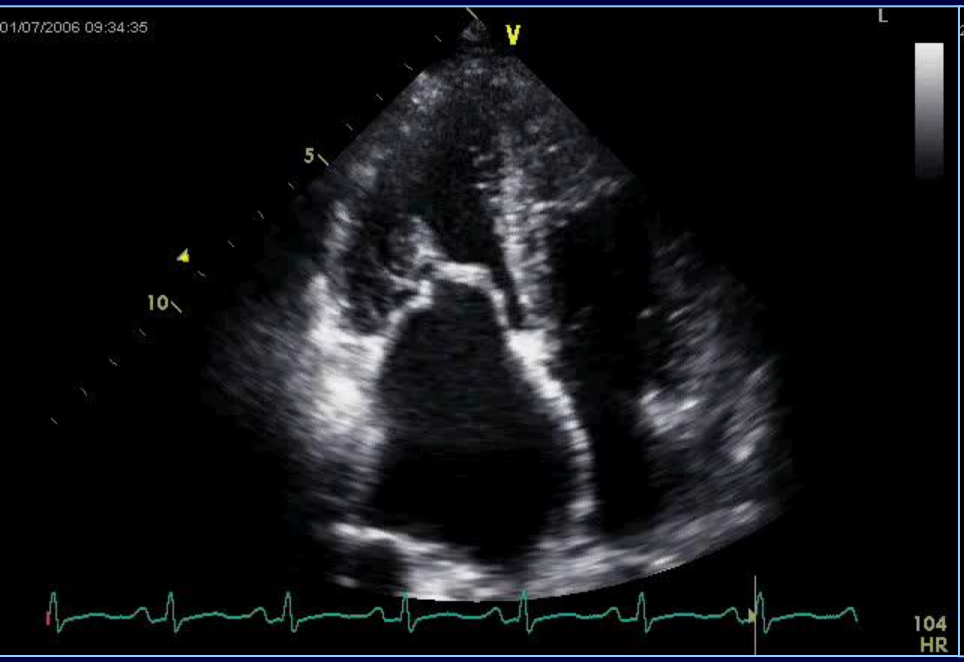


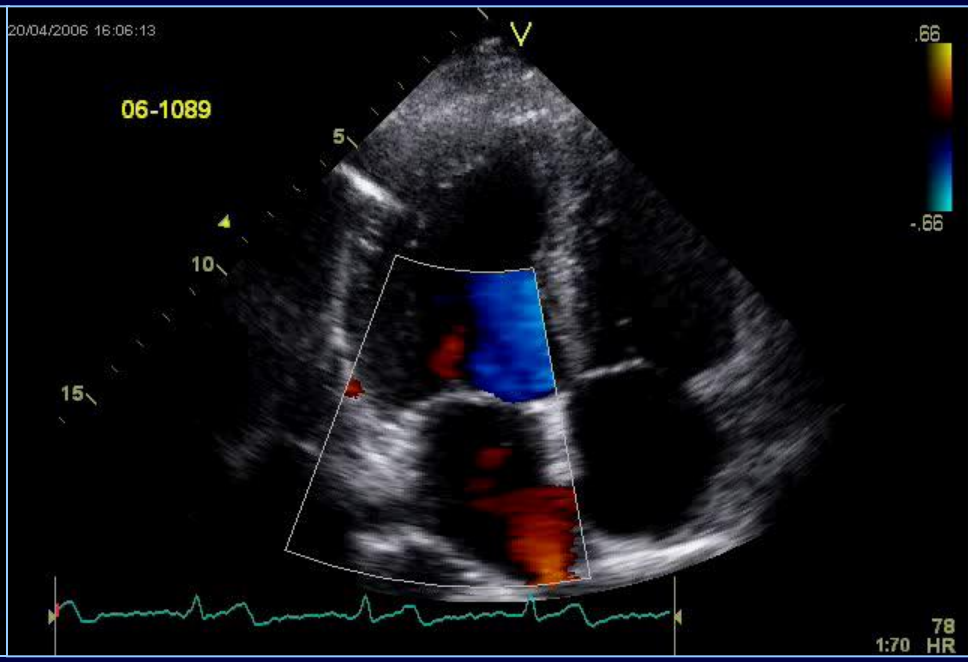
# Interpretation

# Clinical Implication

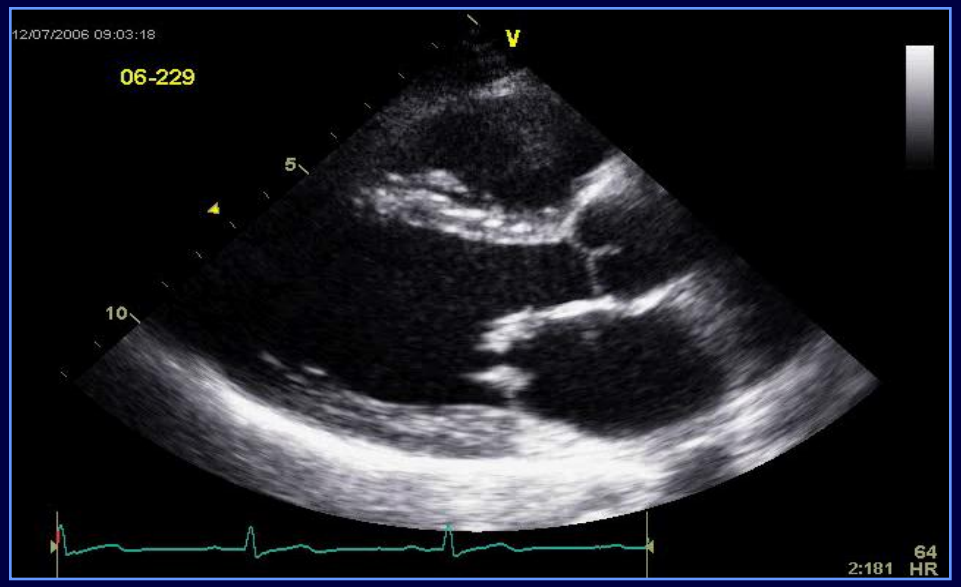

12/07/2006 09:50:23
06-1855
V
5
10
57
3:176 HR

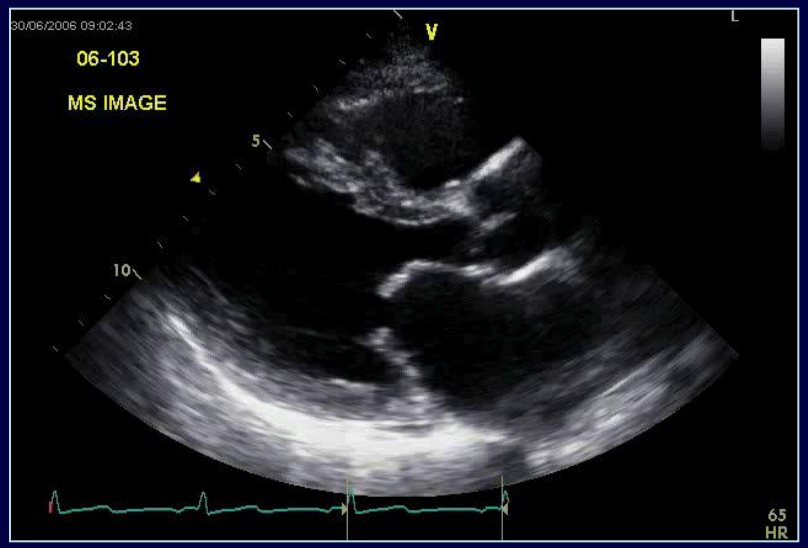

12/07/2006 09:50:39
06-1855
V
.74
-.74
5
10
60
2:94 HR


01/07/2006 09:34:35
V
L
5
10
104
HR

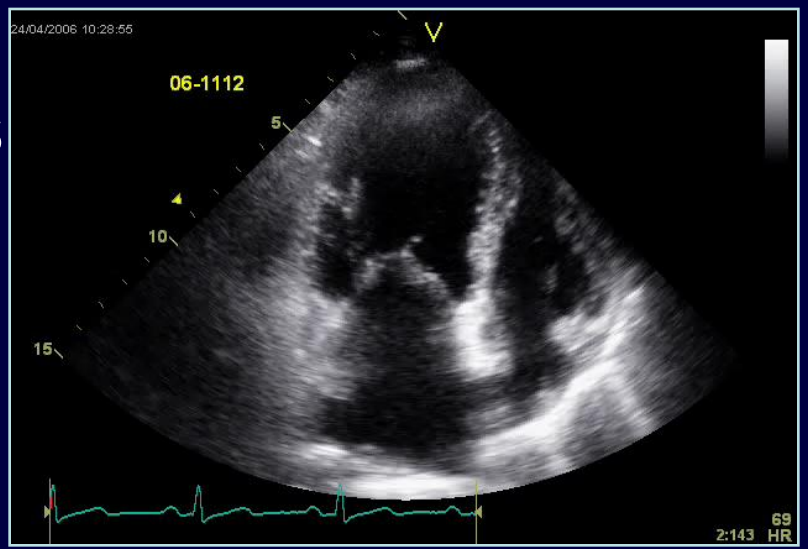

20/04/2006 16:06:13
06-1089
V
.66
-.66
5
10
15
78
1:70 HR

# Mitral Stenosis




- Size of the cardiac chambers
- Estimation of LV function
- Estimation of the PAP
- Associated valvular lesions



# Mitral Stenosis

- The LV diastolic pressure and EF are normal in isolated MS.

- In 25% of pts with isolated MS, EF and other systolic indices below normal

# Mitral Stenosis

- 85% of pts with isolated MS,
  LVEDV: within the normal range
  the remaining pts: reduced LVEDV

- Small, underfilled chamber
  Leftward displacement of the IVS →
  reduction of LV compliance (LV stiffening)

# Mitral Stenosis

- Usually normal or slightly reduced LV mass
- Normal or slightly impaired LV contractility
- Normal elevation of EF and a reduction of ESV during exercise

| | At rest | Exercise |
|---|---|---|
| Mod MS | Normal C.O. | Subnormal increase in C.O. |
| Severe MS | Subnormal C.O. | Declined C.O. |

# Mitral Stenosis

- Exercise testing

  to ascertain the level of physical conditioning

  to elicit covert cardiac symptoms

- Exercise Doppler testing

  exercise hemodynamics

  discrepancy between

  resting echocardiographic findings and the severity of clinical symptoms

# Mitral Stenosis

- Useful parameters on exercise testing

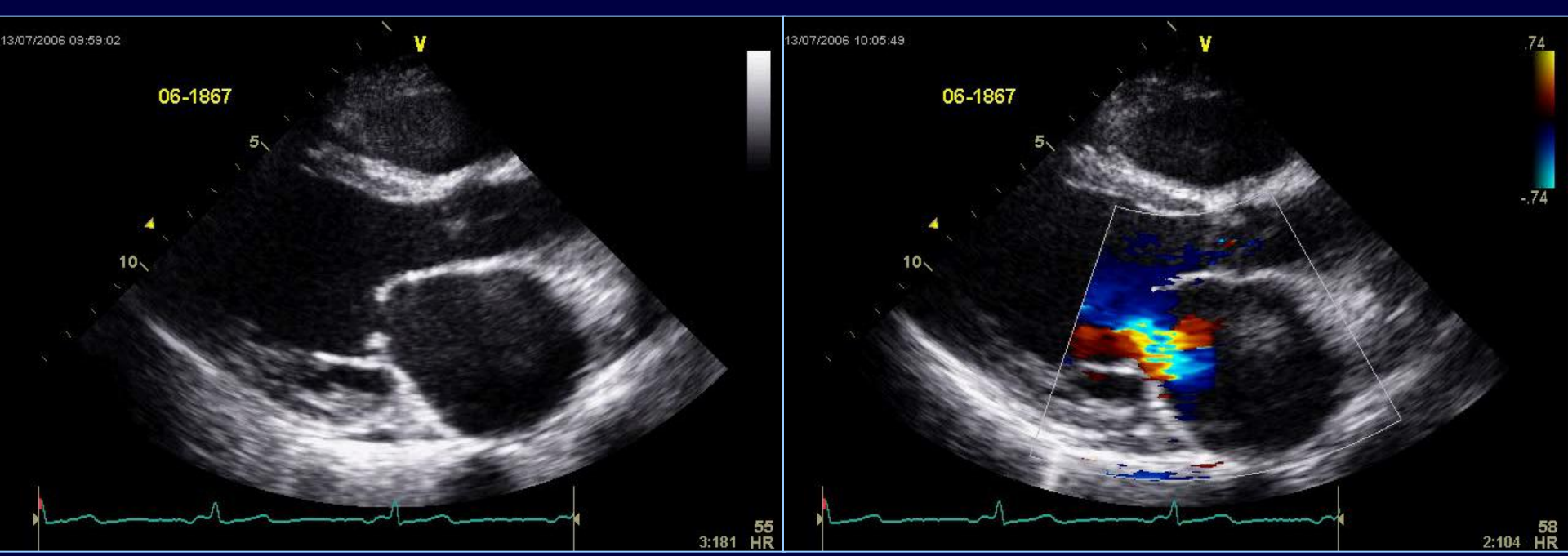
  (1) Exercise duration

  (2) BP and HR response

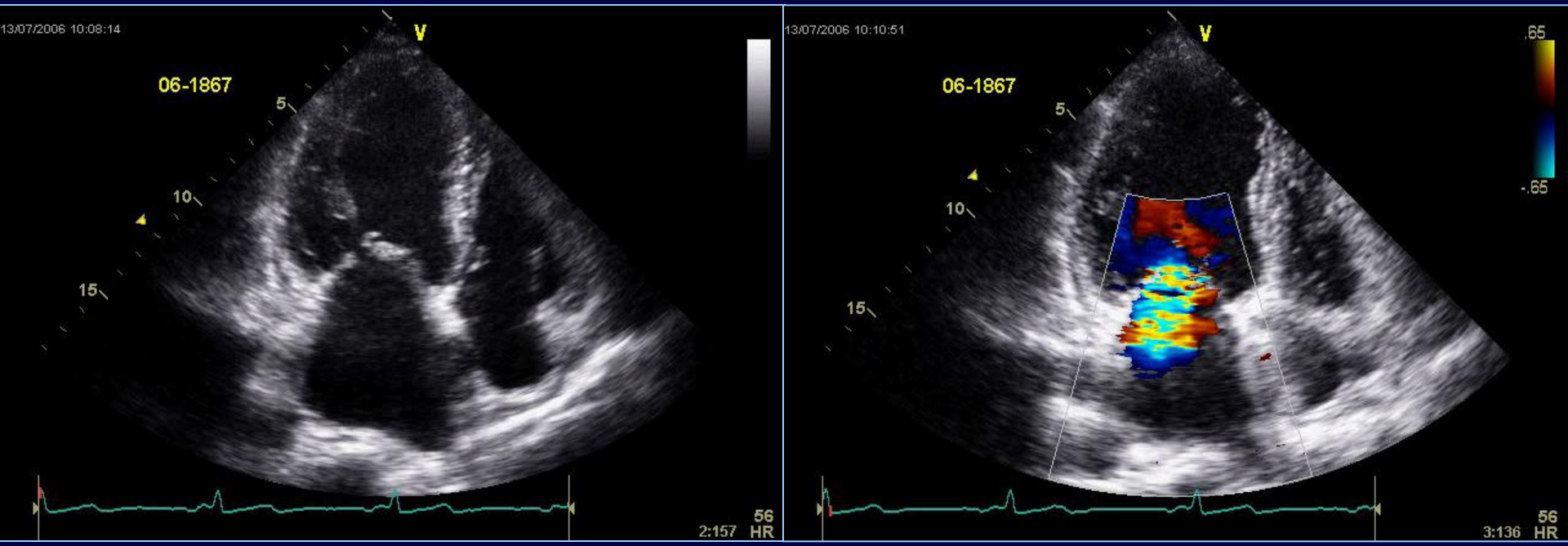
  (3) Change in mean transmitral gradient

  (4) Increase in pulmonary pressures with exercise

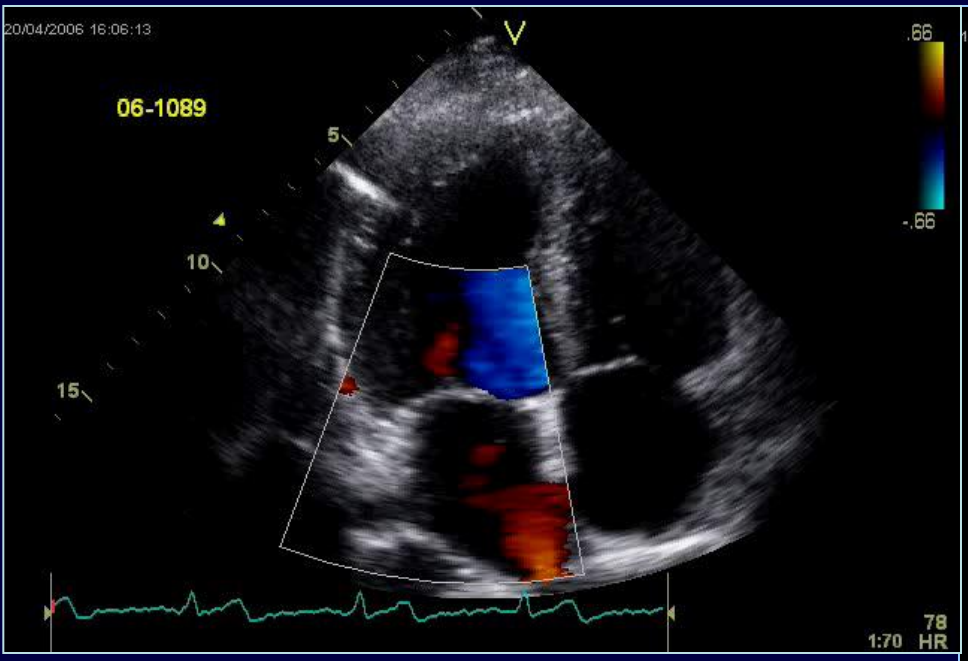
- Exercise PAsP > 60 mmHg: key decision point

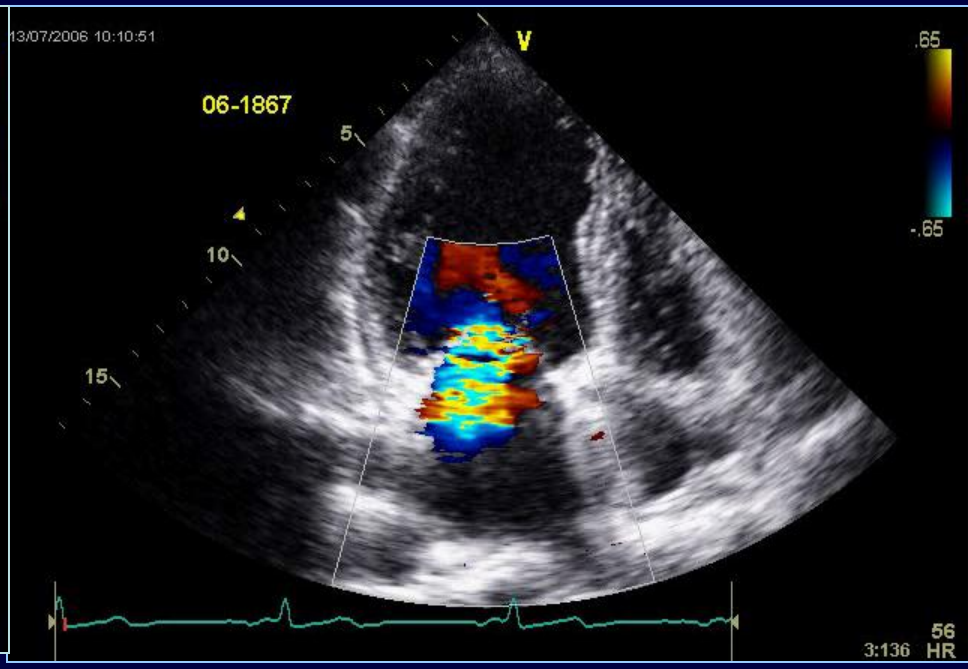



# Echocardiography



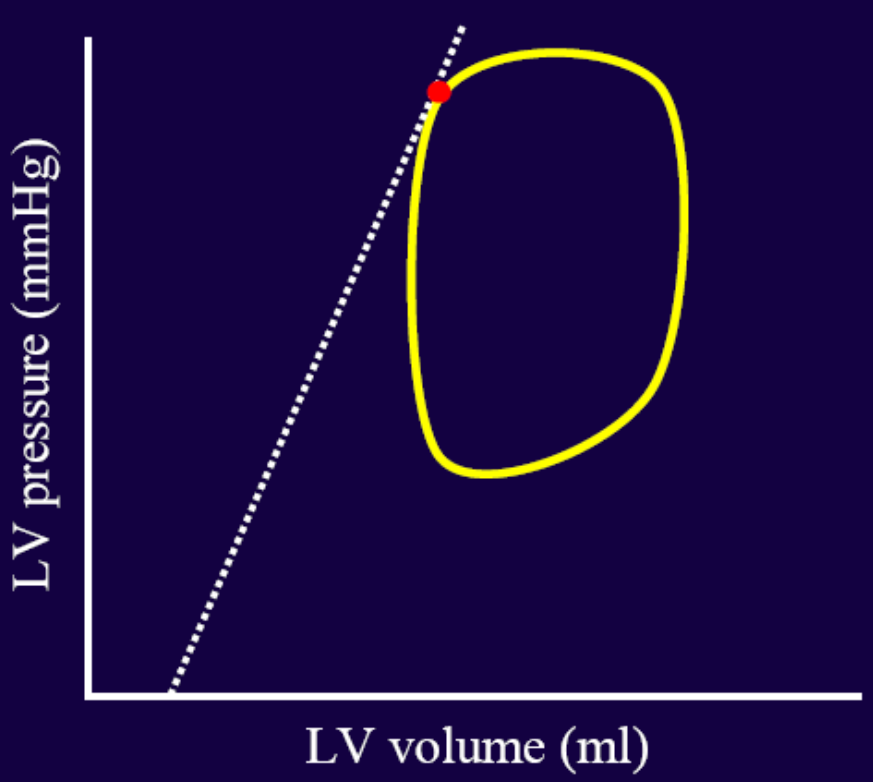
# MR


20/04/2006 16:06:13
06-1089
.66
-.66
78
1:70 HR


13/07/2006 10:10:51
06-1867
.65
-.65
56
3:136 HR

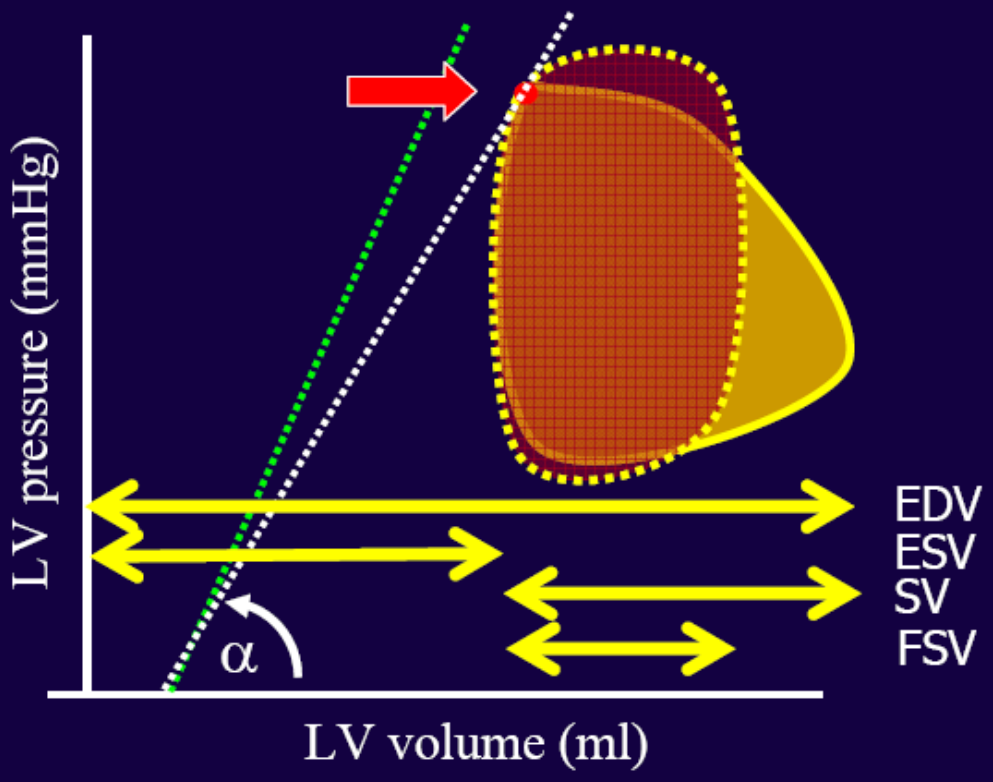
# Assessment of LV function

## *End-Systolic Elastance*




**Normal**

EF = SV/EDV




**Severe MR**

FEF = FSV/EDV

# CHRONIC MR

***Natural History***

- "MR begets MR"
- Chronic volume overload → LV dysfunction
- Ejection phase indices >> LV contractility
- Symptoms are subtle and late

# Mitral Regurgitation

- Reduced resistance to LV emptying
  (LV afterload)
- Reduction in LV size during systole
- **The initial compensation:**
  **more complete LV emptying**

- LV volume increases progressively with time

# Mitral Regurgitation

- LV compliance is often increased.

- Since EF rises in severe MR in the presence of normal LV function, even a modest reduction in this parameter (<60%) reflects significant dysfunction.

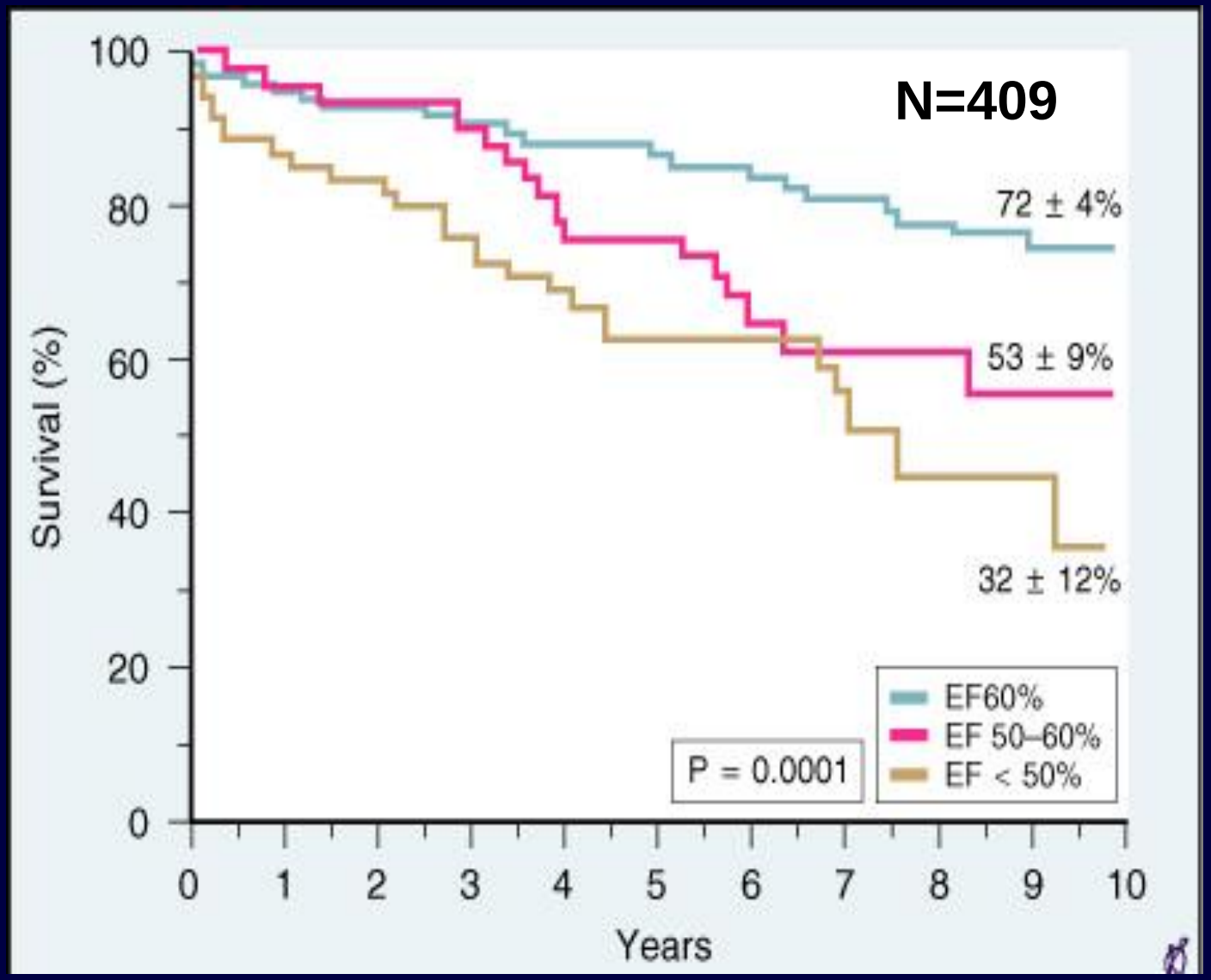
# Mitral Regurgitation

- Practical clinical measures for early contractile dysfunction

  LV end systolic dimension $\geq$ 45 mm

  EF $\leq$ 60%

# Late survival after operation of MR

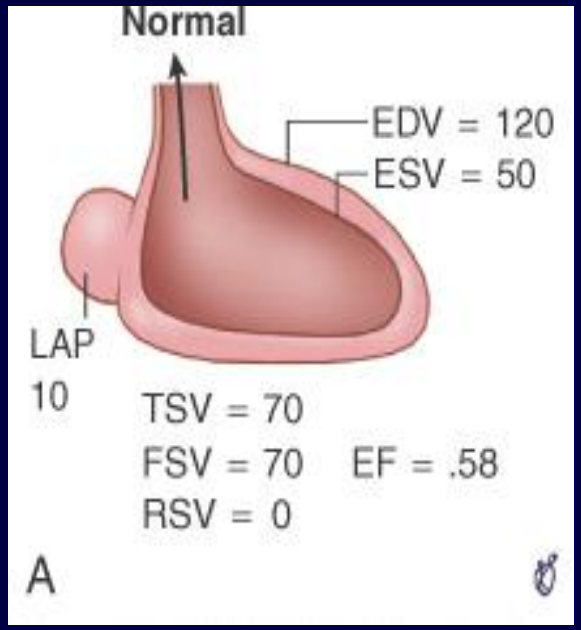


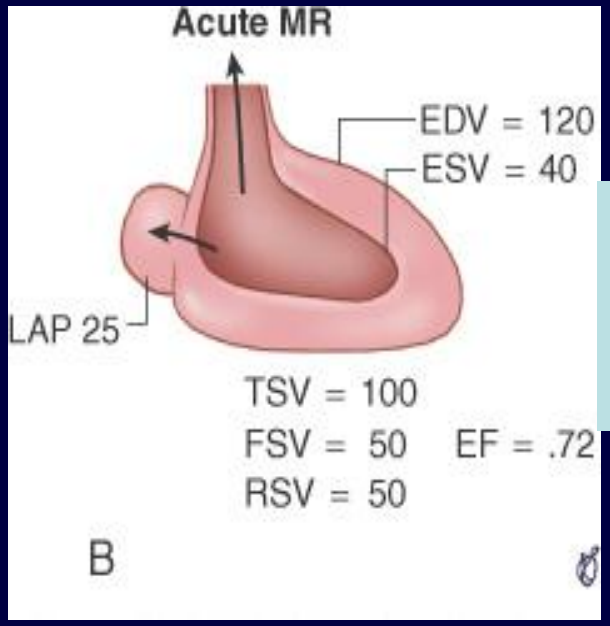

Enriquez-Sarano, Circulation 1994;90:830

# Operation is Indicated for

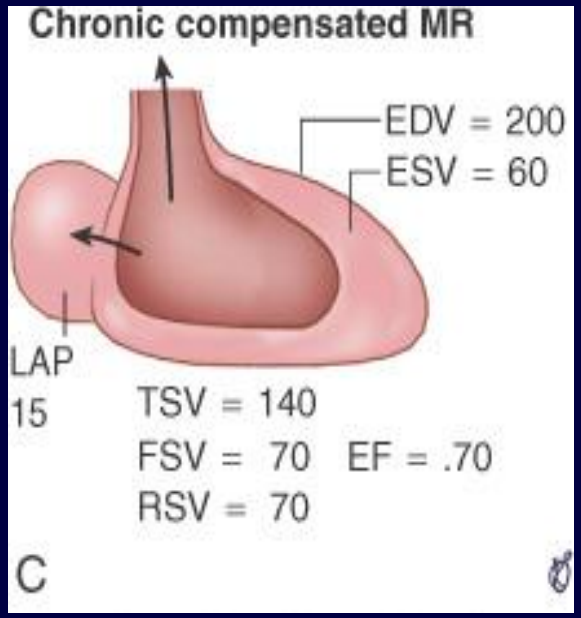
- Most pts with **severe MR and any symptoms**
- **Asymptomatic** pts with **chronic severe** MR who demonstrate **mild to moderate** LV dysfunction (**EF: 0.30 - 0.60 & ESD: 40 - 55 mm**)
- The patient with **severe LV dysfunction (EF < 0.30 and/or ESD > 55 mm)** poses a higher risk but may undergo surgery if chordal preservation is likely.

# Three phases of MR

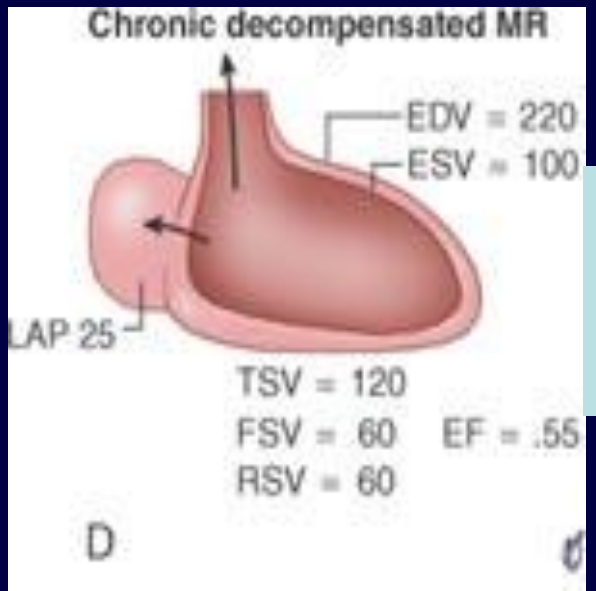



ESV ↓
LAP ↑
EF




LAP ↓
EDV ↑
EF




EF ↓
LAP ↑
EDV

# Mitral Regurgitation

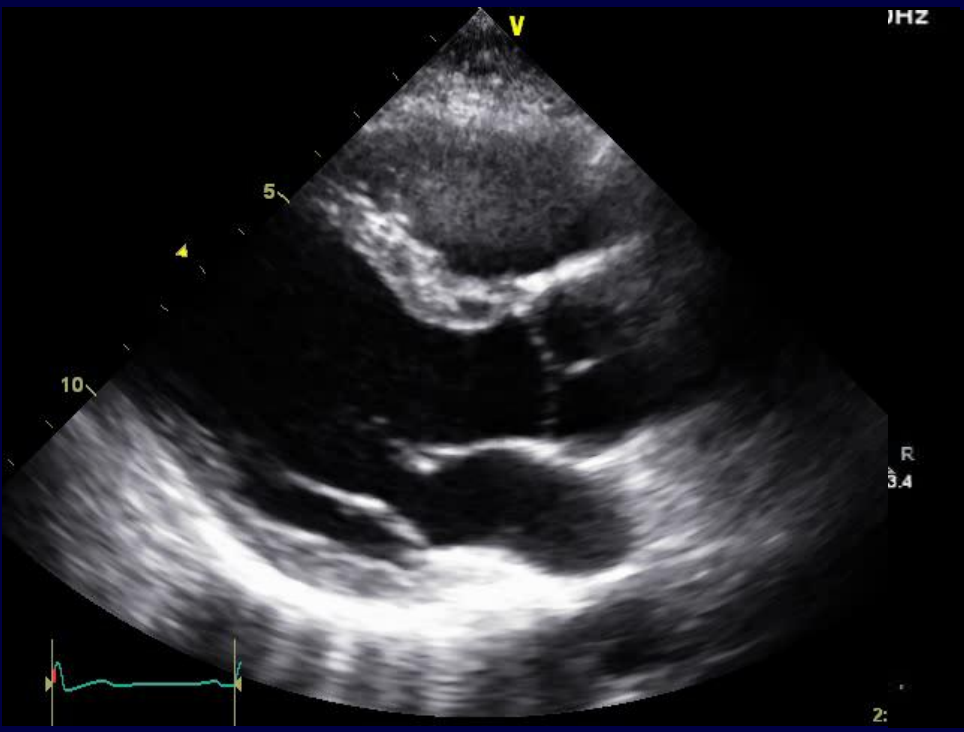
- With decompensation,
  increased chamber stiffness
  raising the diastolic pr at any volume

- End-systolic pressure/volume relation:
  a useful index for evaluating LV function

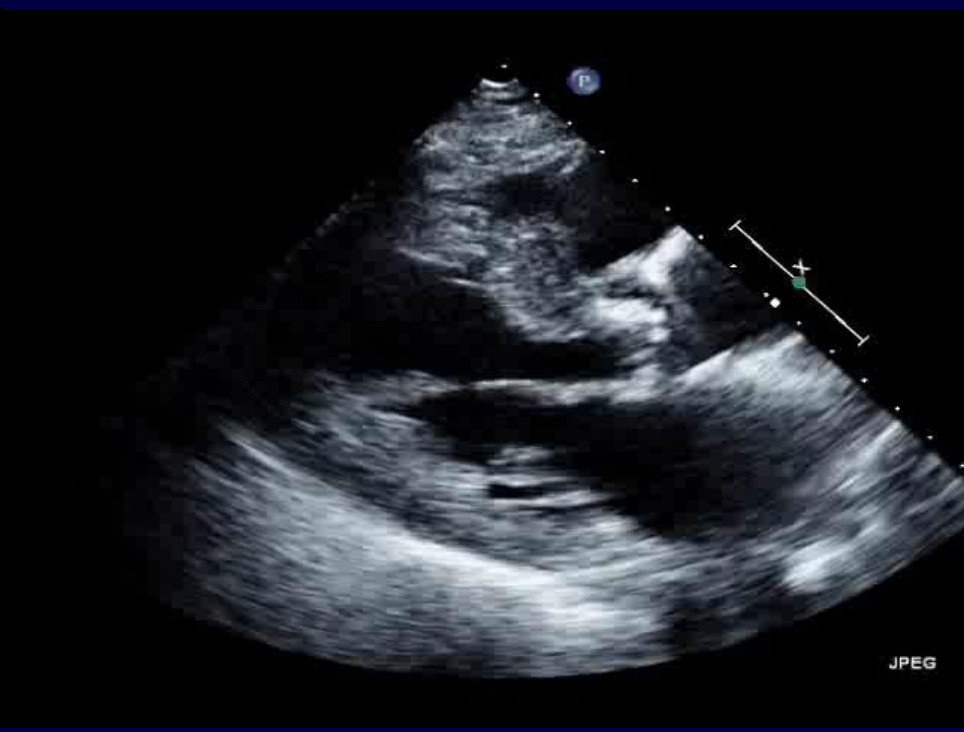
- Preoperative LVESD > 40 mm
  high likelihood of impaired LV systolic function following surgery

# 2D Echocardiography

**Normal**




**Aortic Stenosis**



# Aortic Stenosis

- The obstruction to LV outflow
- Chronic pressure overload
- Concentric LVH
- Increased wall thickness

  → normalization of wall stress (afterload)

  → maintain LV contractile function

# Aortic Stenosis

- Increased myocardial cell mass and increased interstitial fibrosis
  → diastolic dysfunction

- Sustain a large PG across the AV for many years without a reduction in CO, LV dilation, or the development of symptoms

# Aortic Stenosis

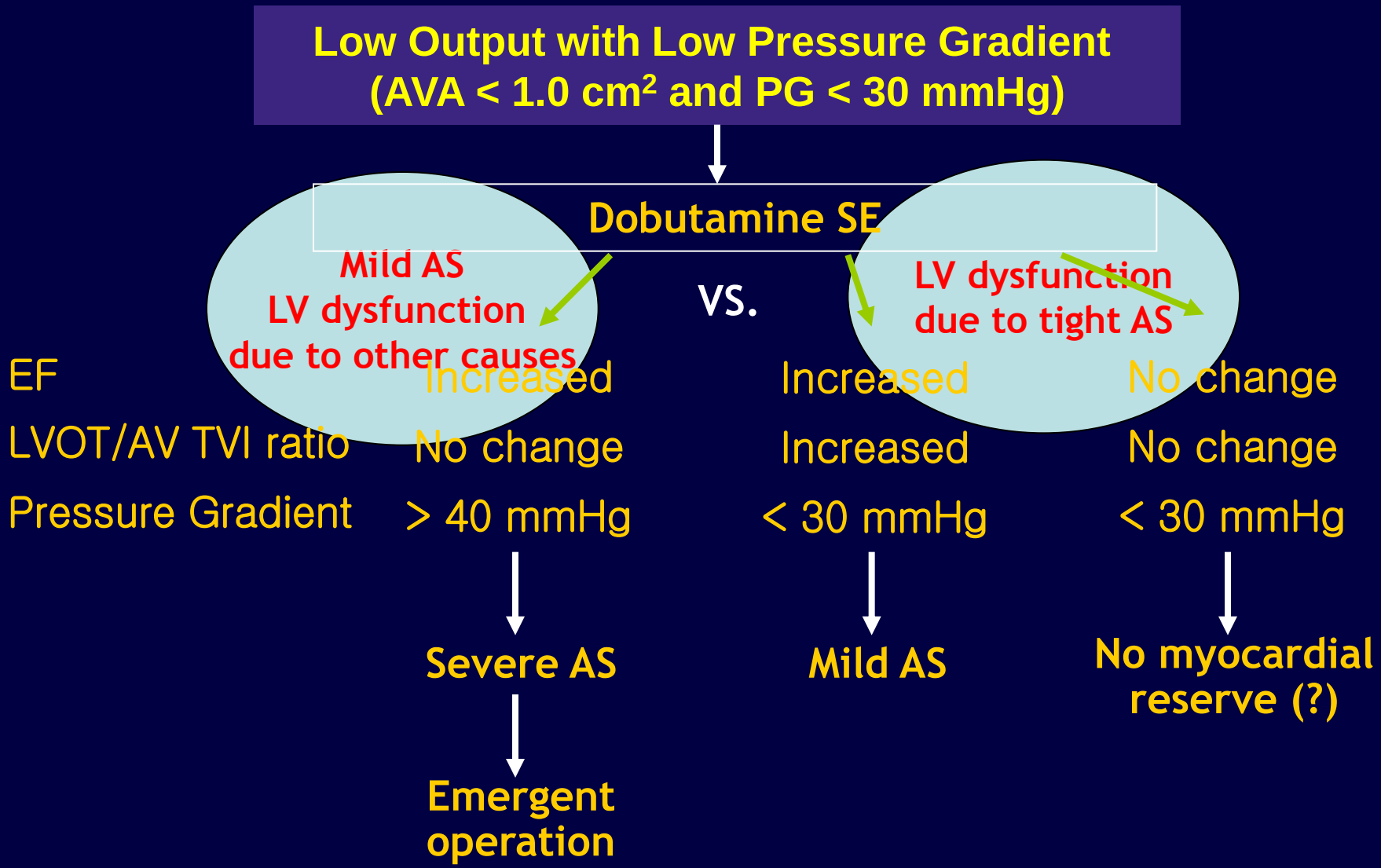
- Excessive hypertrophy becomes maladaptive
- LV dilatation and reduced systolic shortening reflect impairment of LV function.
- The elevated LVEDP signifies the presence of LV dilatation and diminished compliance

# Aortic Stenosis

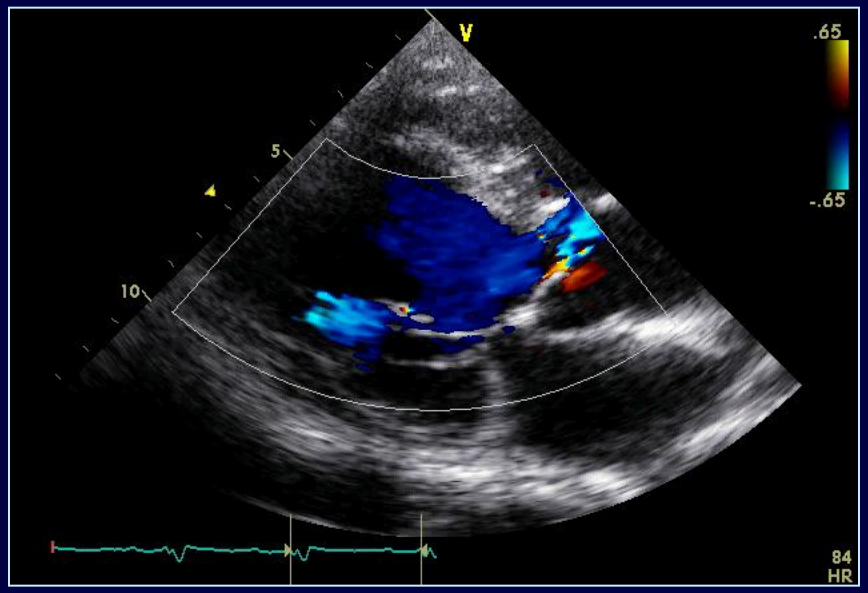
- CO fails to rise normally during exercise in severe AS

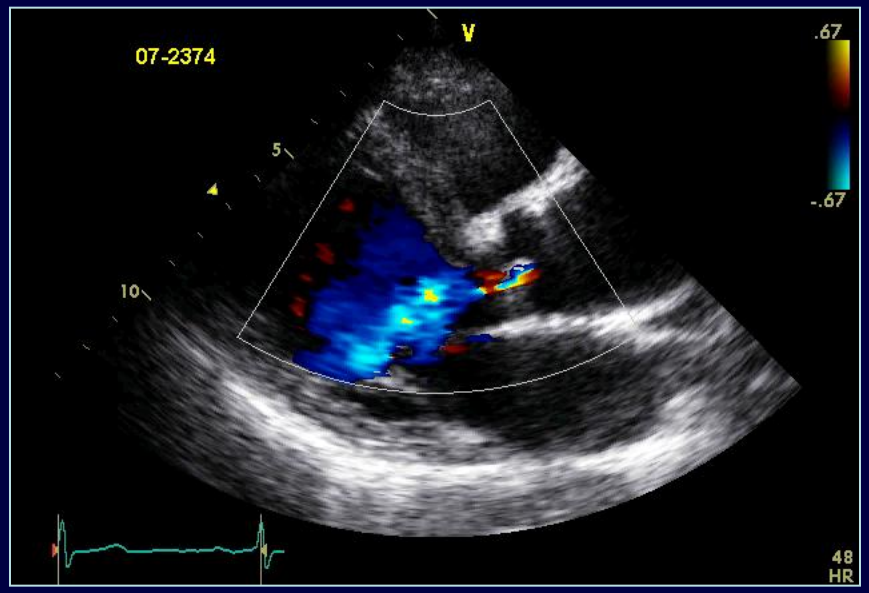
- Late in the course,
  the CO and LV–aortic PG decline,
  and the mean LA, PA and RV pressures rise.

# AS with Low Pressure Gradient



# Aortic Regurgitation

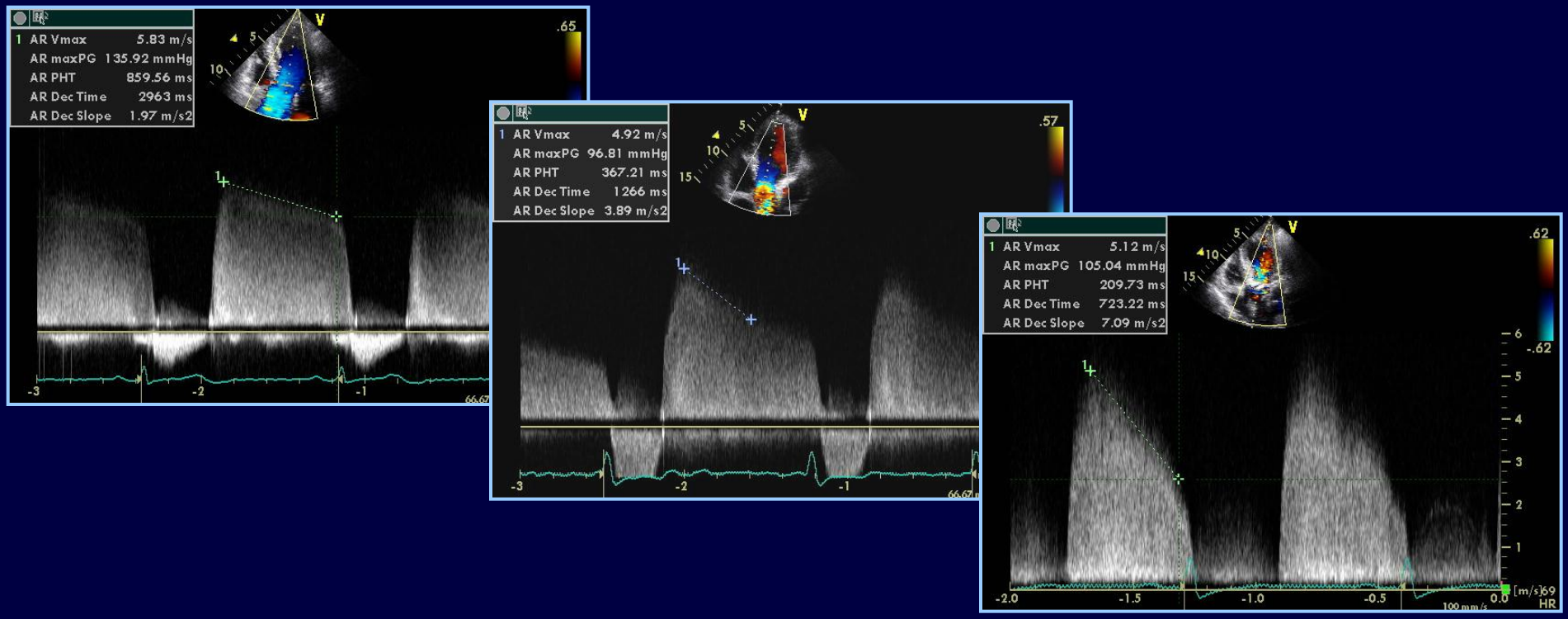



- Increased total SV ejected by the LV
- The entire LV SV is ejected into a high-pressure zone, the aorta.

# Continuous Wave Doppler

## CW Doppler Signal Density




**Qualitative**

**Overlap between moderate and severe AR**

**Aortic compliance, Blood pressure, LV size and compliance, etc**

**An indicator of acuity rather than severity**

# Aortic Regurgitation

- An increase in the LVEDV (increased preload): the major hemodynamic compensation

- The dilatation and eccentric LVH allow the LV to eject a larger SV without requiring any increase in the relative shortening of each myofibril.

# Aortic Regurgitation

- Severe AR may occur with a normal effective forward SV and a normal LV EF, together with an elevated LVEDP and EDV.

- Chronic AR permits the LV to function as an effective high-compliance pump, handling a large SV, often with little increase in filling pressure.

# Aortic Regurgitation

- Chronic AR: LV preload and afterload are both increased.
- LV systolic function is maintained through the combination of chamber dilation and hypertrophy.
- As LV function deteriorates, the LVEDV rises further and the forward SV and EF decline.

# Aortic Regurgitation

- In advanced stages of decompensation, LA, PA wedge, PA, RV, and RA pressures rise and the effective (forward) CO falls, at first during exercise and then at rest.

- As the LV decompensates, interstitial fibrosis increases, compliance declines, and LVEDP and EDV rise.

# Take Home Message

- LVEDV, LV mass and contractility is usually normal in isolated MS.
- **More complete LV emptying** is the initial compensation to reduced LV afterload in MR.
- **EF rises in severe MR** in the presence of normal LV function.

# Take Home Message

- **LVH is an adaptive mechanism** and maintains LV contractile function **in AS**.
- An **increase in the LVEDV** is the major hemodynamic compensation in **AR**.
- Eccentric LVH allow the LV to eject a larger SV without requiring any increase in contractility.