

# Diastology 2011

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**Disclosures: None**





**ASE**

**American Society of  
Echocardiography**

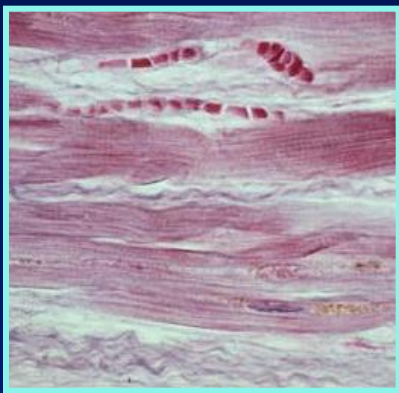
*Heart & Circulation  
Ultrasound Specialists*

**Is *EVERYBODY* a member!?!**

ΕΡΡΟΣ ΕΚΤΡΟΦΟΛΟΓΙΑΣ  
ΑΝΕΡΓΩΝ ΕΤΕΡΩΝ ΕΤΕΡΩΝ

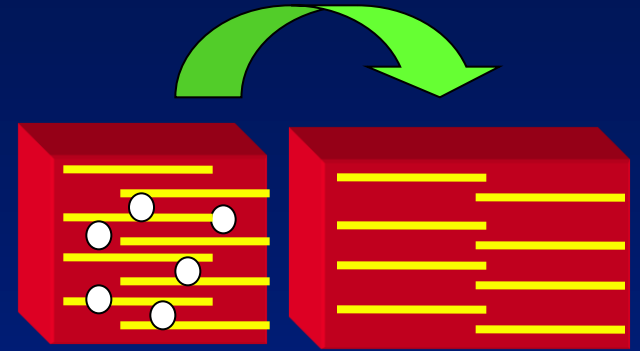
# Systolic and Diastolic Function

## *Myocardium to Ventricle*



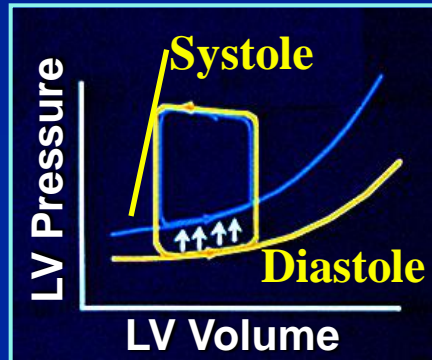
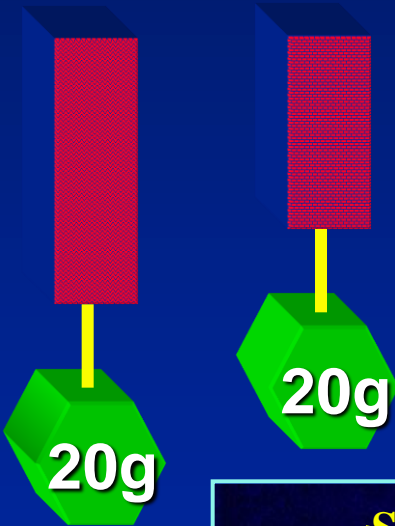
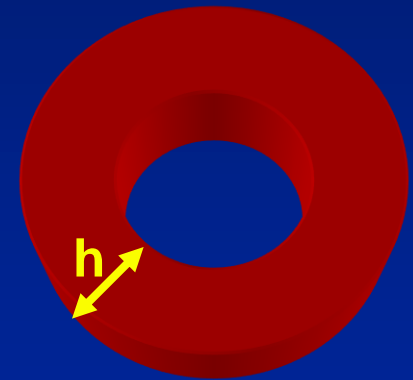
Tissue  
Elastance

Activation



Stress/strain  
Relationship

Geometry

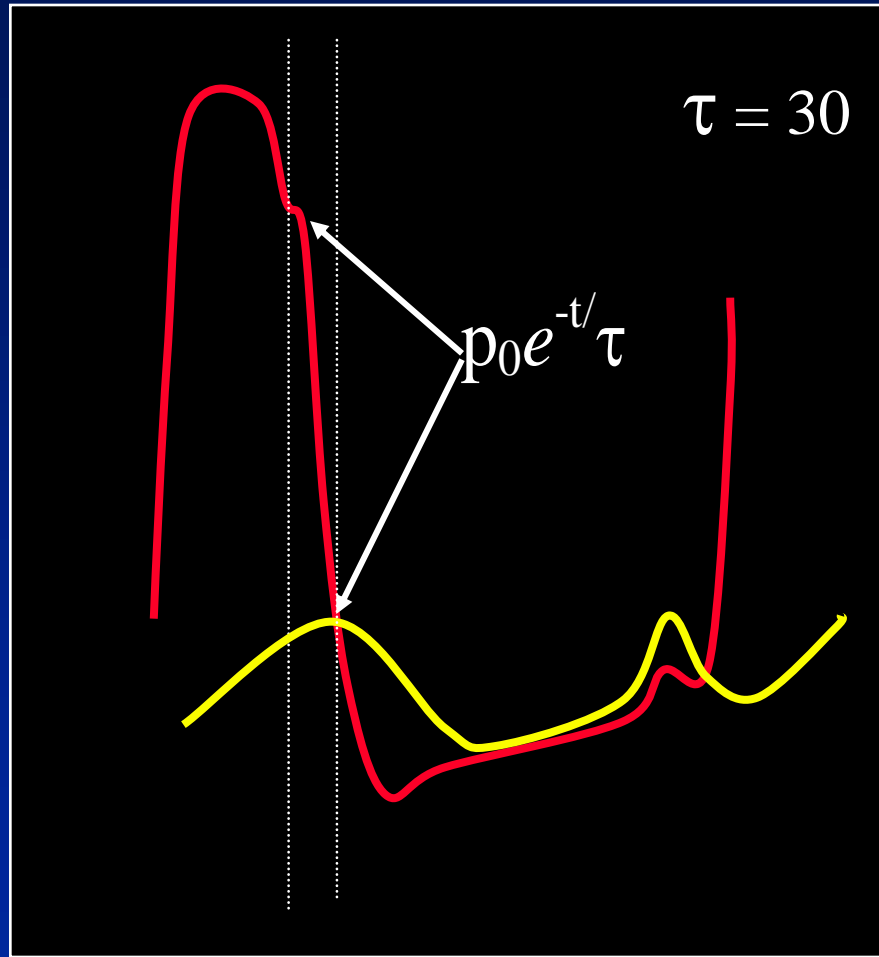


Pressure-Volume  
Curve



# LV Relaxation

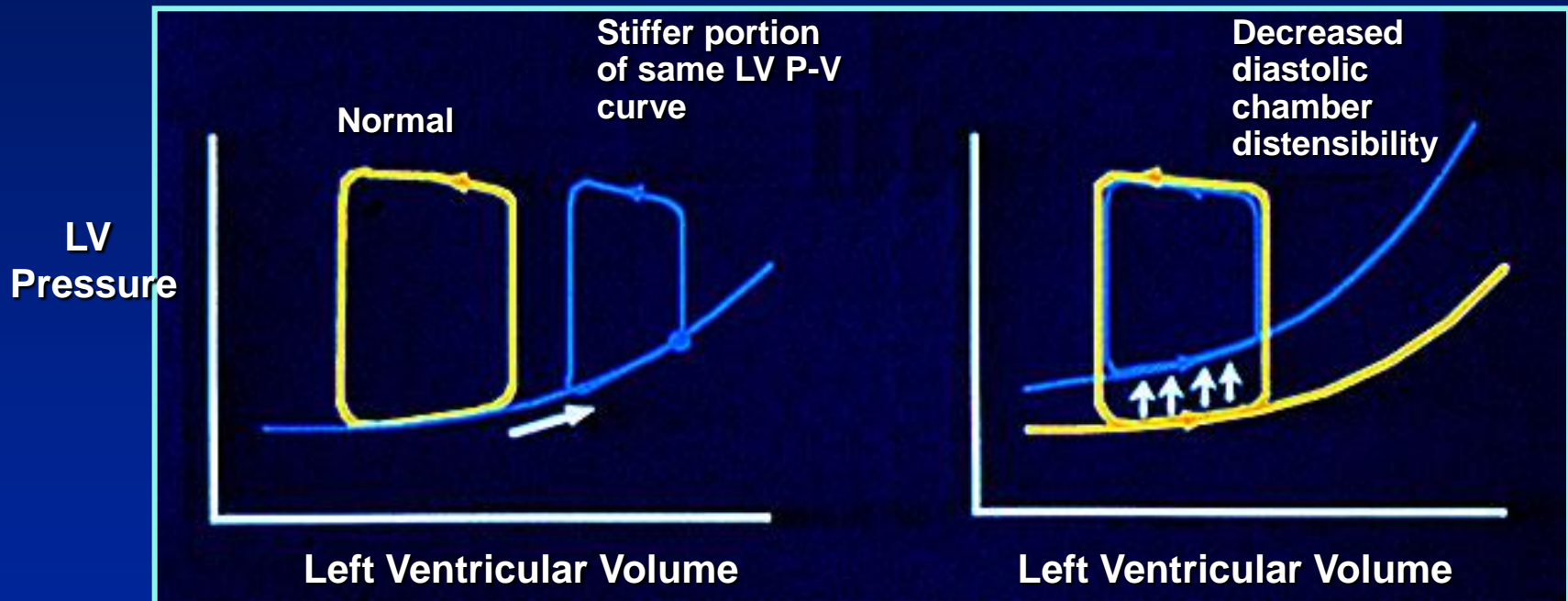
## *Exponential Pressure Decay in IVR*



# LV Pressures-Volume Curves

## Systolic Failure

## Diastolic Failure



Lorell BH. Ann Rev Med 1991;42:411-37



# Passive Elasticity

<u>Component</u>	<u>Contribution</u>
Collagen	++++
Titin	++++
Actin	++
Intermediate	+
Microtubules	-

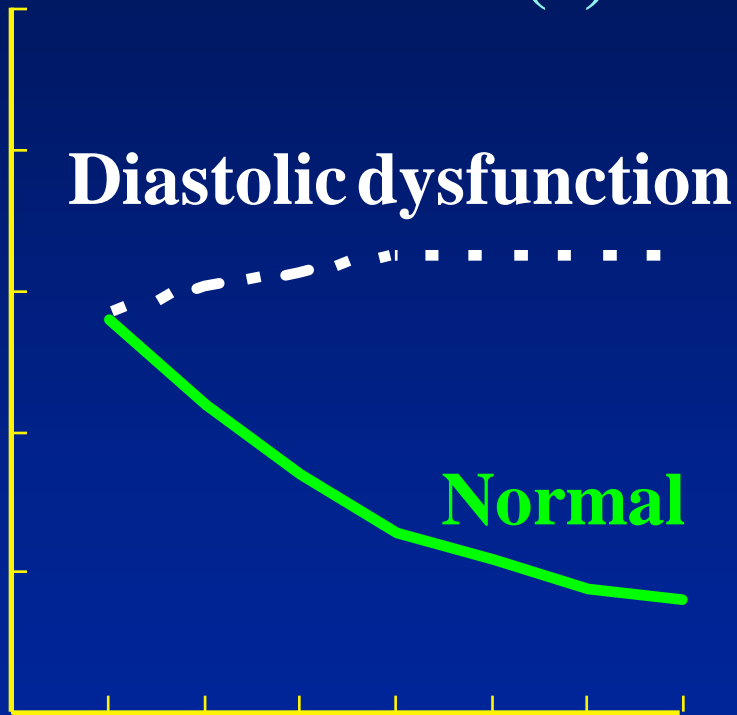
Passive Tension in Cardiac Muscle: Contribution of Collagen, Titin, Microtubules, and Intermediate Filaments

Granzier HL, Irving TC      Biophysical Journal 1995; 68: 1027-1044



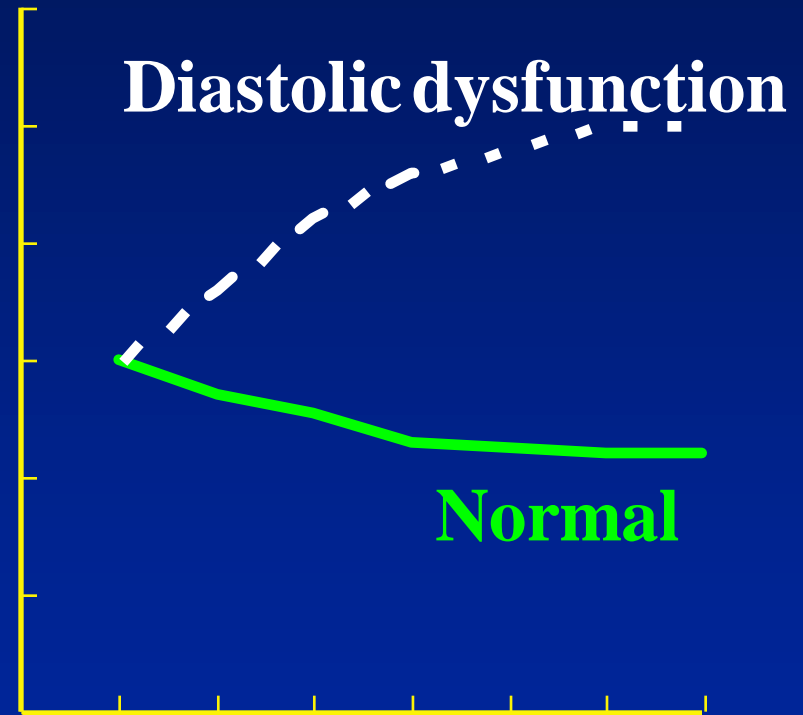
# Exercise Can Unmask Delayed Relaxation

Relaxation Time  
Constant ( $\tau$ )



Heart Rate (bpm)

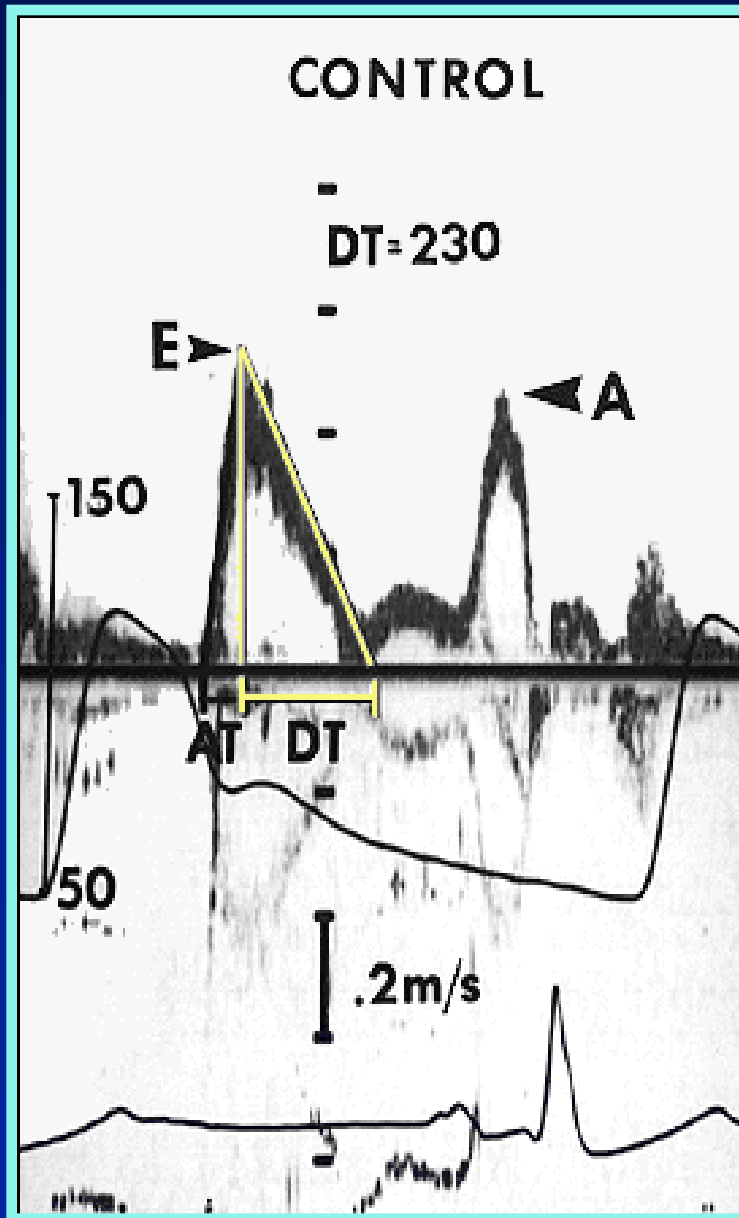
LVEDP



Heart Rate (bpm)



# What Determines Transmitral Flow?

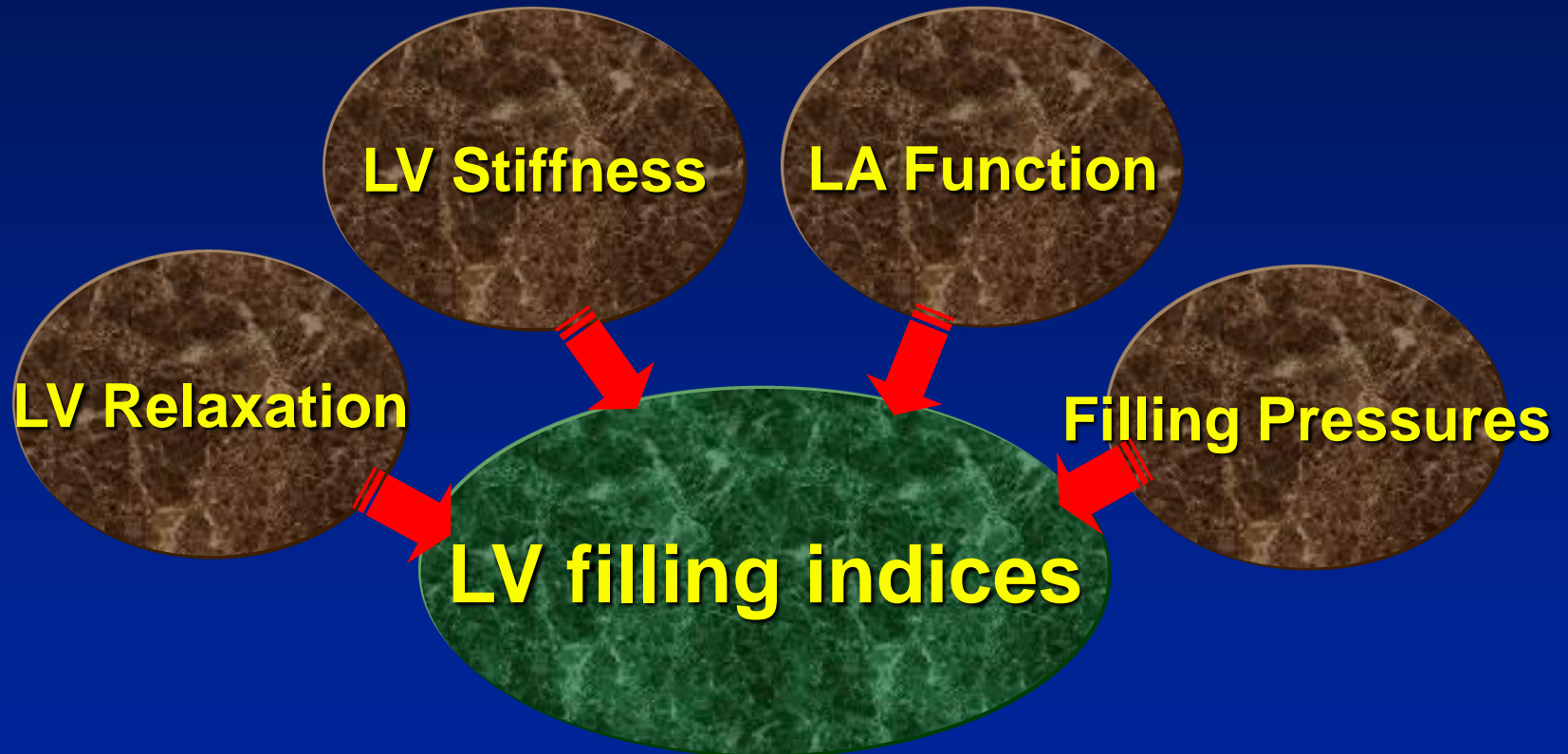


*Key parameters of the mitral inflow pattern:*

- E
- E VTI
- AT
- DT
- A
- A VTI
- A Duration
- E/A



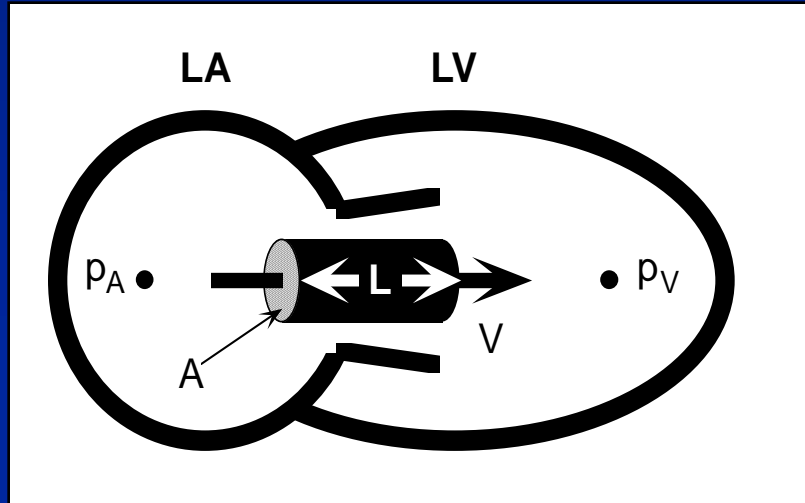
# Determinants of Diastolic Function



# Transmitral Flow

## *Physical Determinants*

- *In physical terms, we can only speak of factors that **accelerate** flow and factors that **decelerate** flow*
- *Acceleration is governed by the magnitude of the transmitral gradient  $\Rightarrow$  **relaxation***
- *Deceleration is governed by the equilibration of LA and LV pressure  $\Rightarrow$  **compliance***



# Transmitral Flow

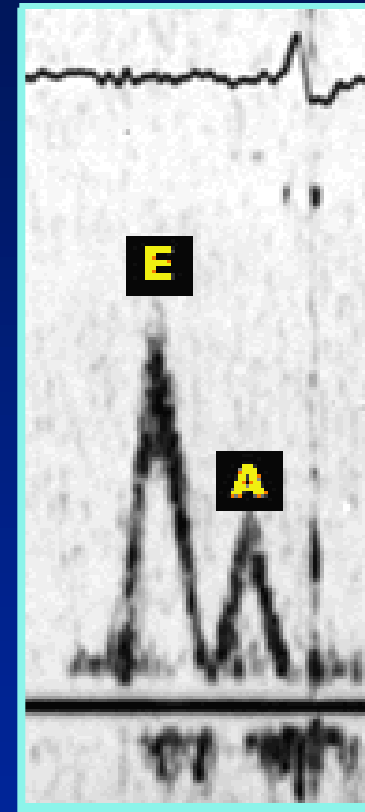
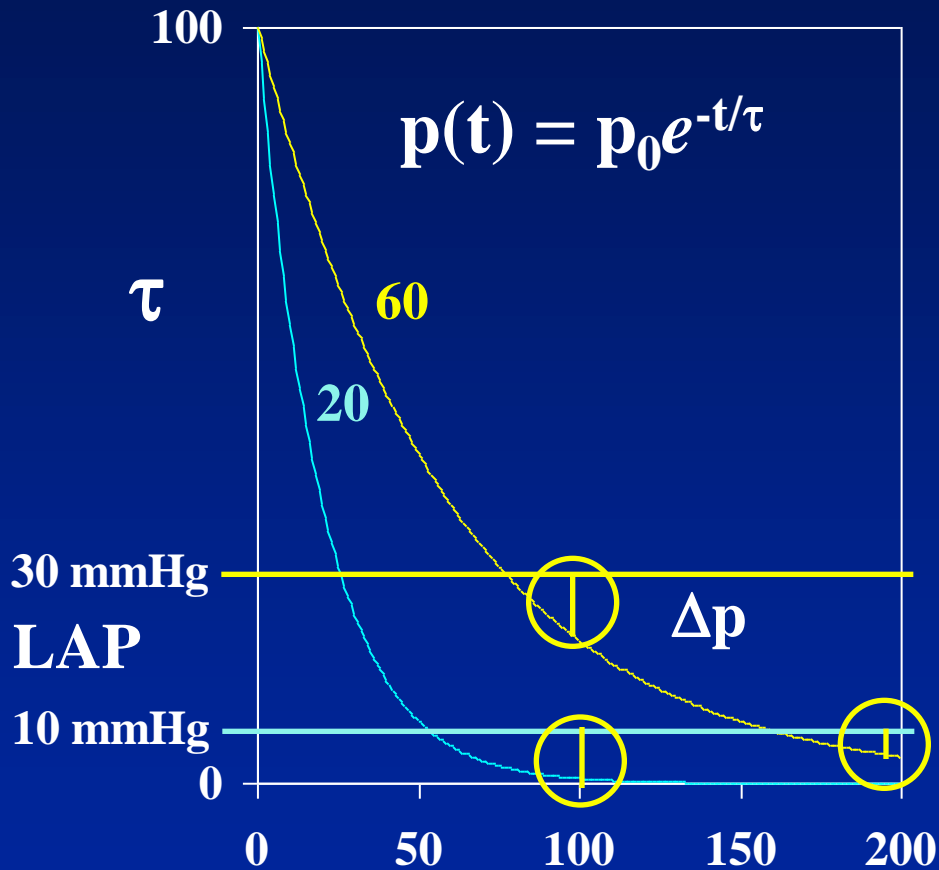
## *Physical Determinants of Acceleration*

- *By Newton's second law,  $a = F/m$*
- *$m =$  mitral inertance, the mass of blood being accelerated across the mitral valve*
  - $\approx 3 \times$  valve diameter (reduced in mitral stenosis)
- *$F =$  transmitral pressure difference*
  - **Grows as LV pressure drops exponentially below left atrium**
  - $p(t) = p_0 e^{-t/\tau}$
  - $dp/dt = -p/\tau = p_{LA}/\tau$



# MV Acceleration

*Proportional to  $p_{LA}/\tau$*

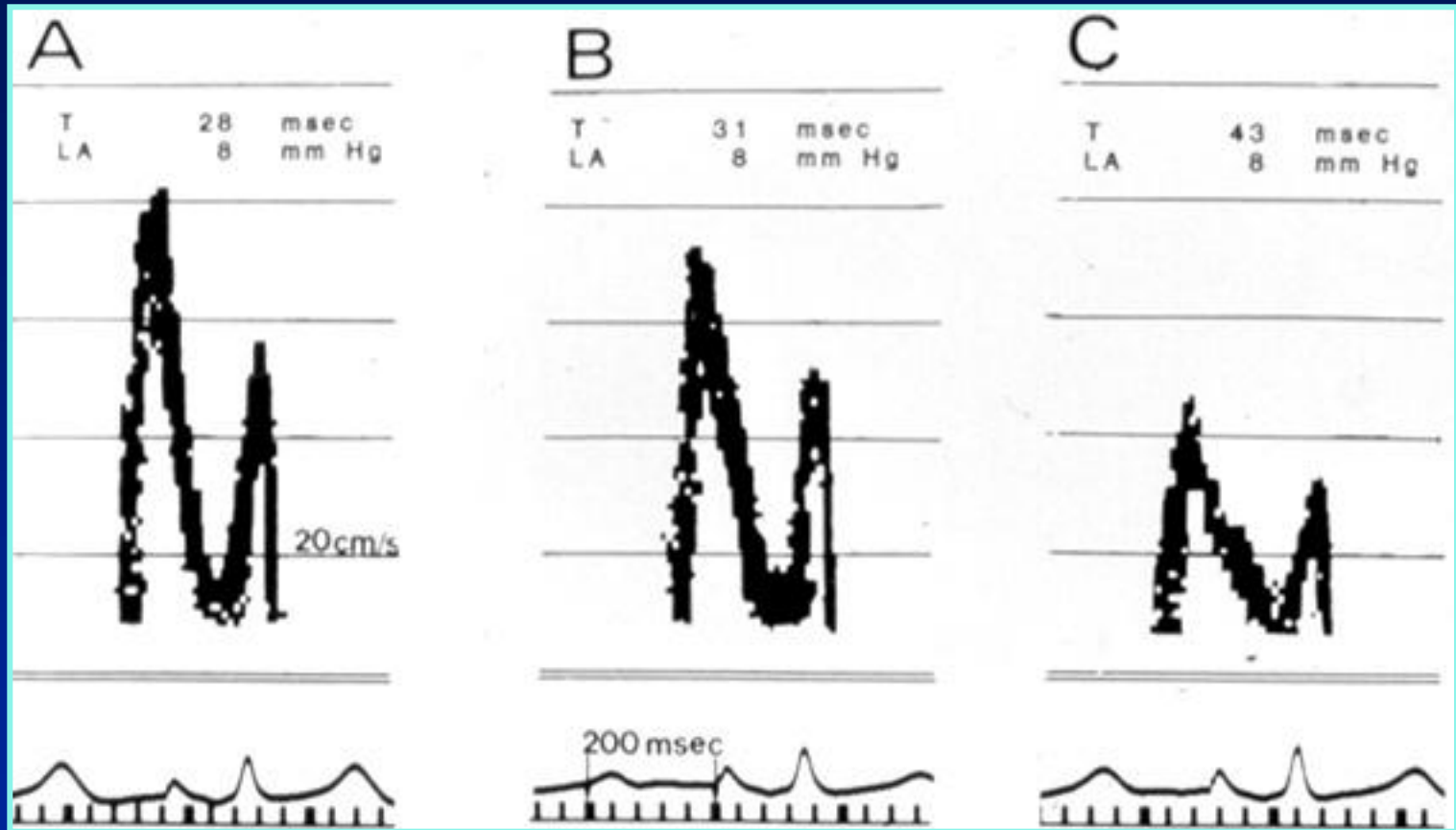


*Doppler Echocardiography*

Rate of rise is proportional to growth of transmitral pressure gradient,  $\Delta p$ , and  $d\Delta p/dt = LAP/\tau$

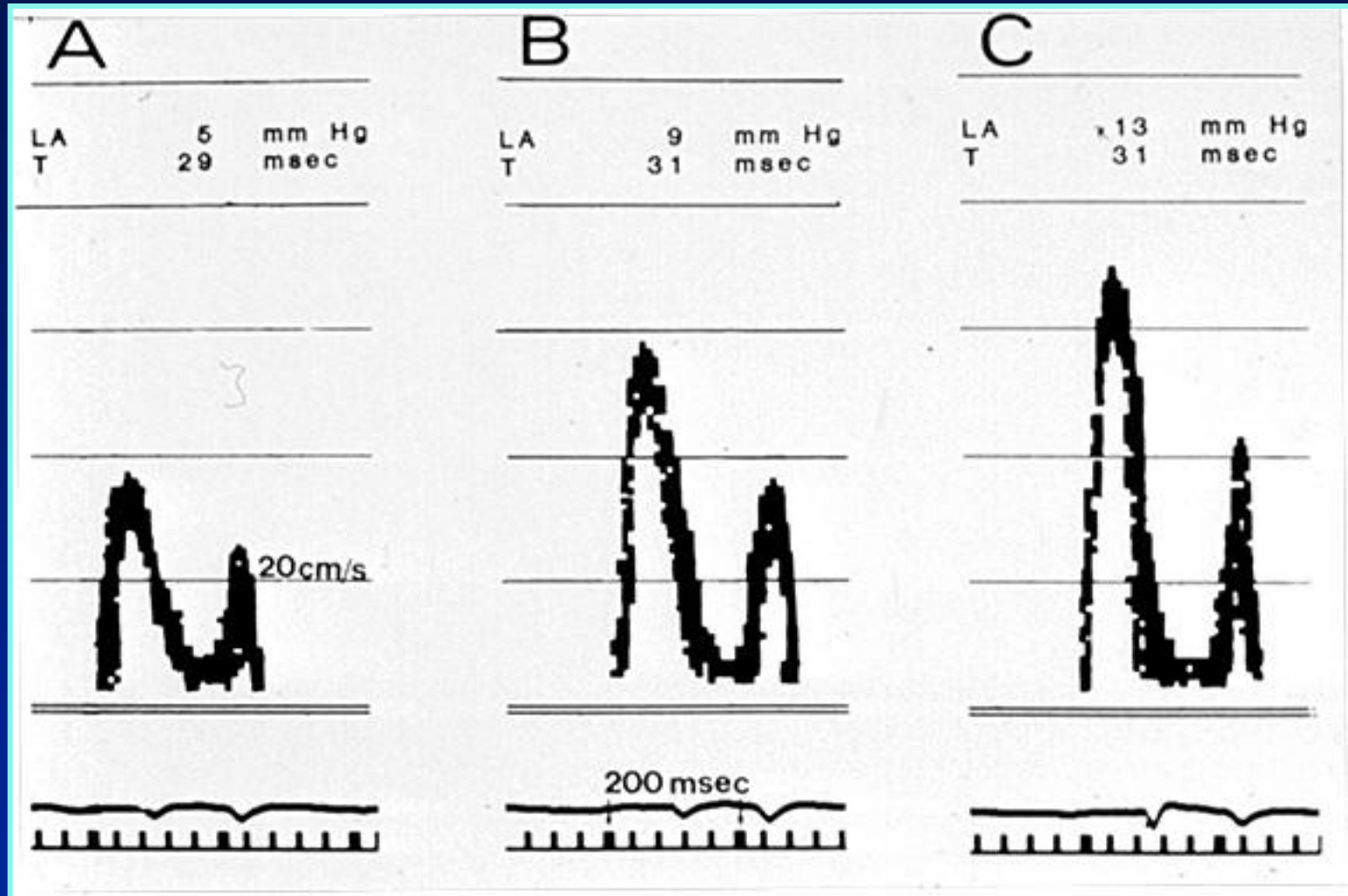


# Effect of Relaxation on LV Inflow



With delayed relaxation, acceleration is slowed and E peak is lower.

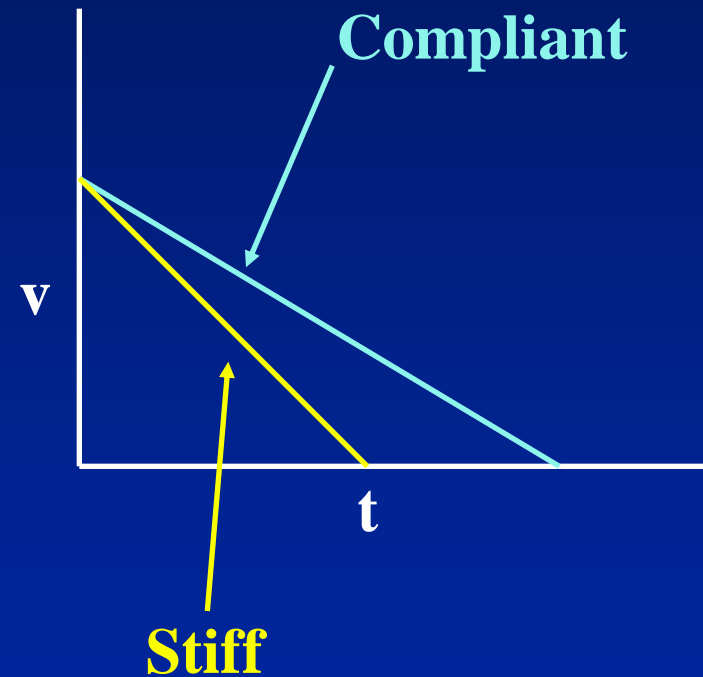
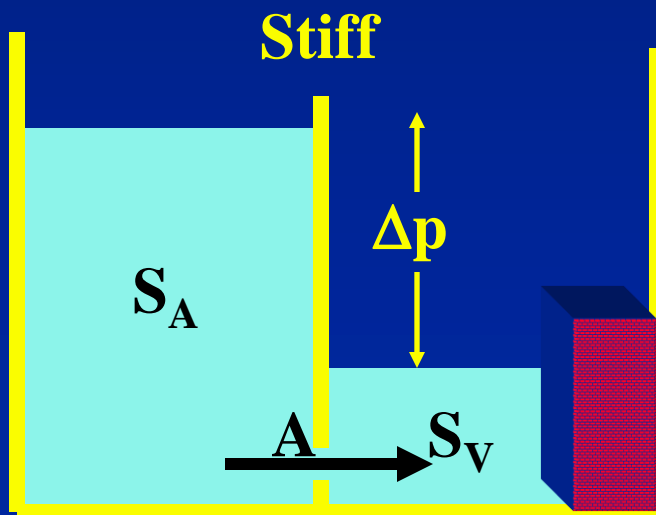
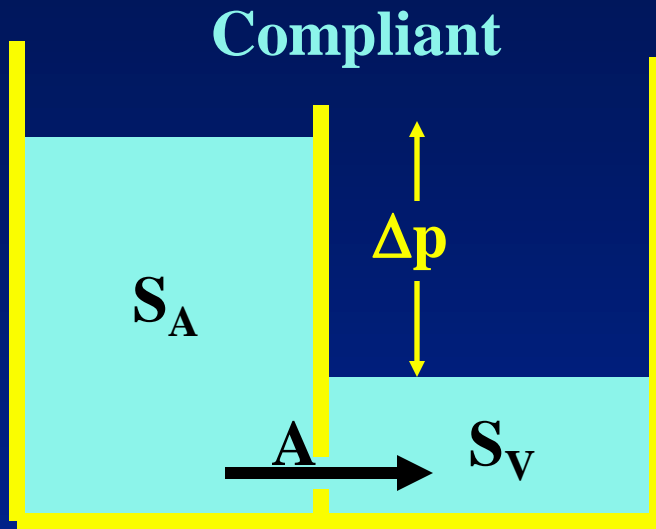
# Effect of LAP on LV Inflow



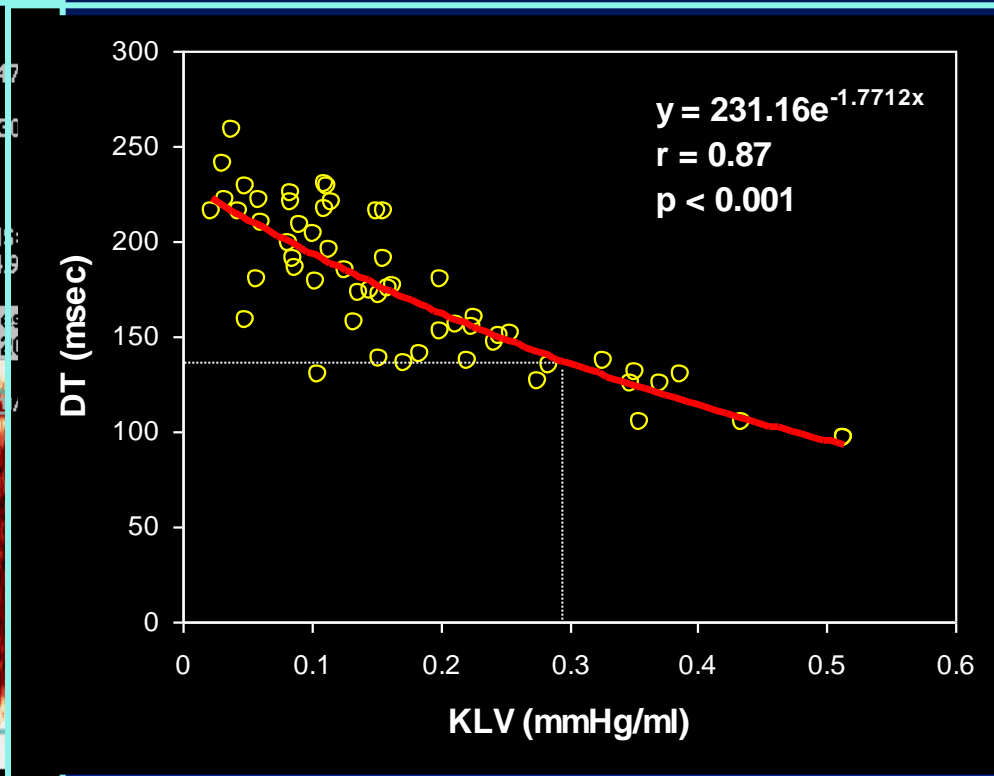
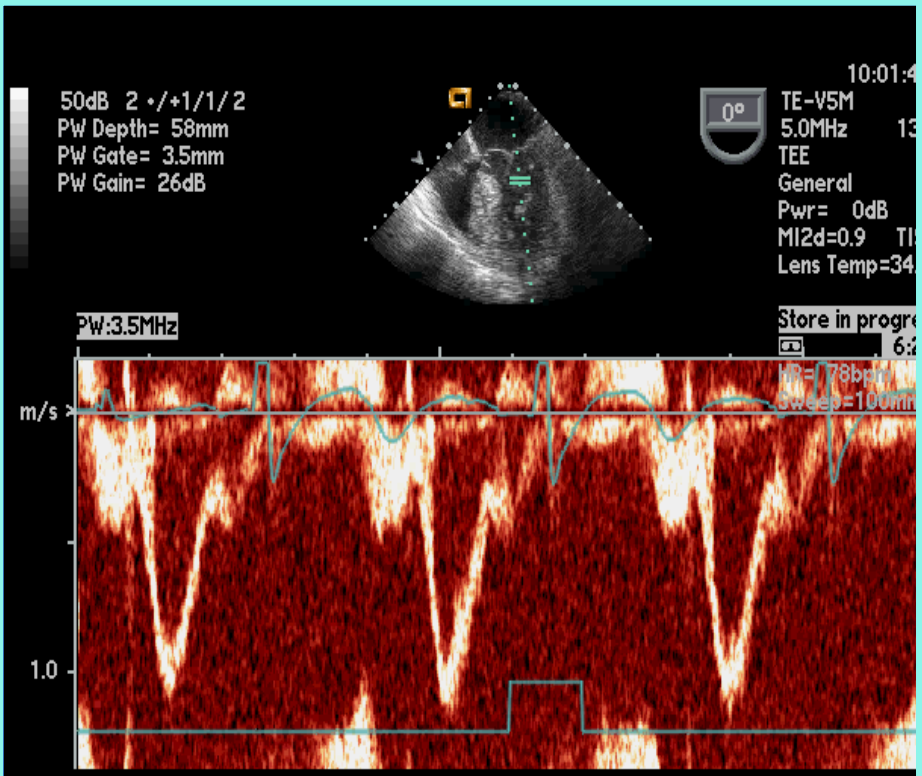
With rising LA pressure, acceleration is faster and E peak is higher.

# Physical Determinants of Deceleration

*Stiffer Ventricle = Shorter Decel Time*



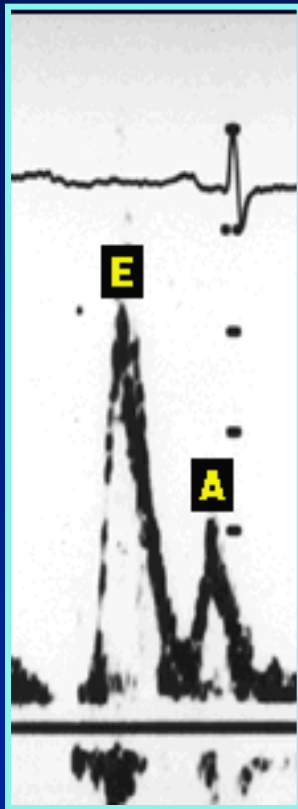
# LV stiffness ?



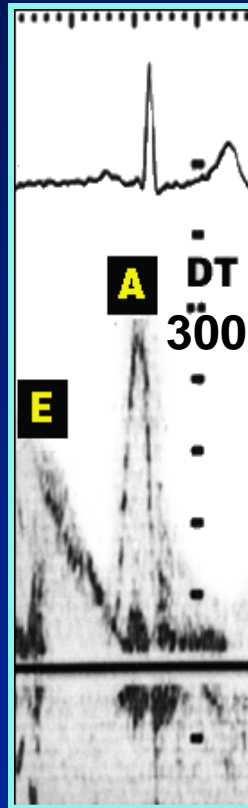
- DT = 140 msec

Answer = operating stiffness (dp/dV)  
increased ( $\cong 0.30$ mmHg/ml)

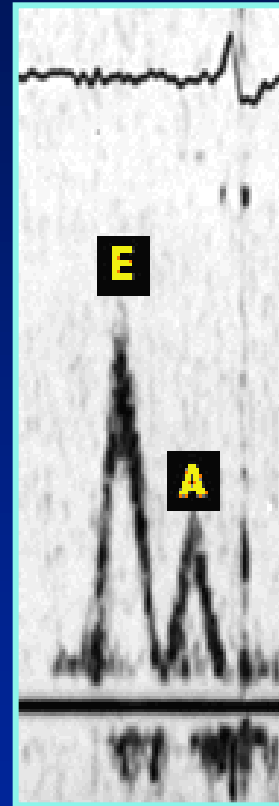
# Left Ventricular Filling Patterns



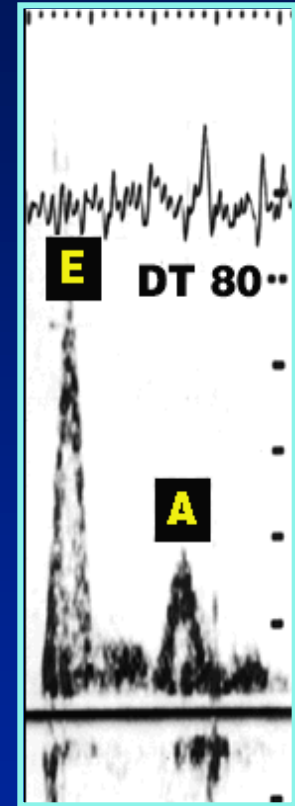
*Normal*



*Delayed  
relaxation*



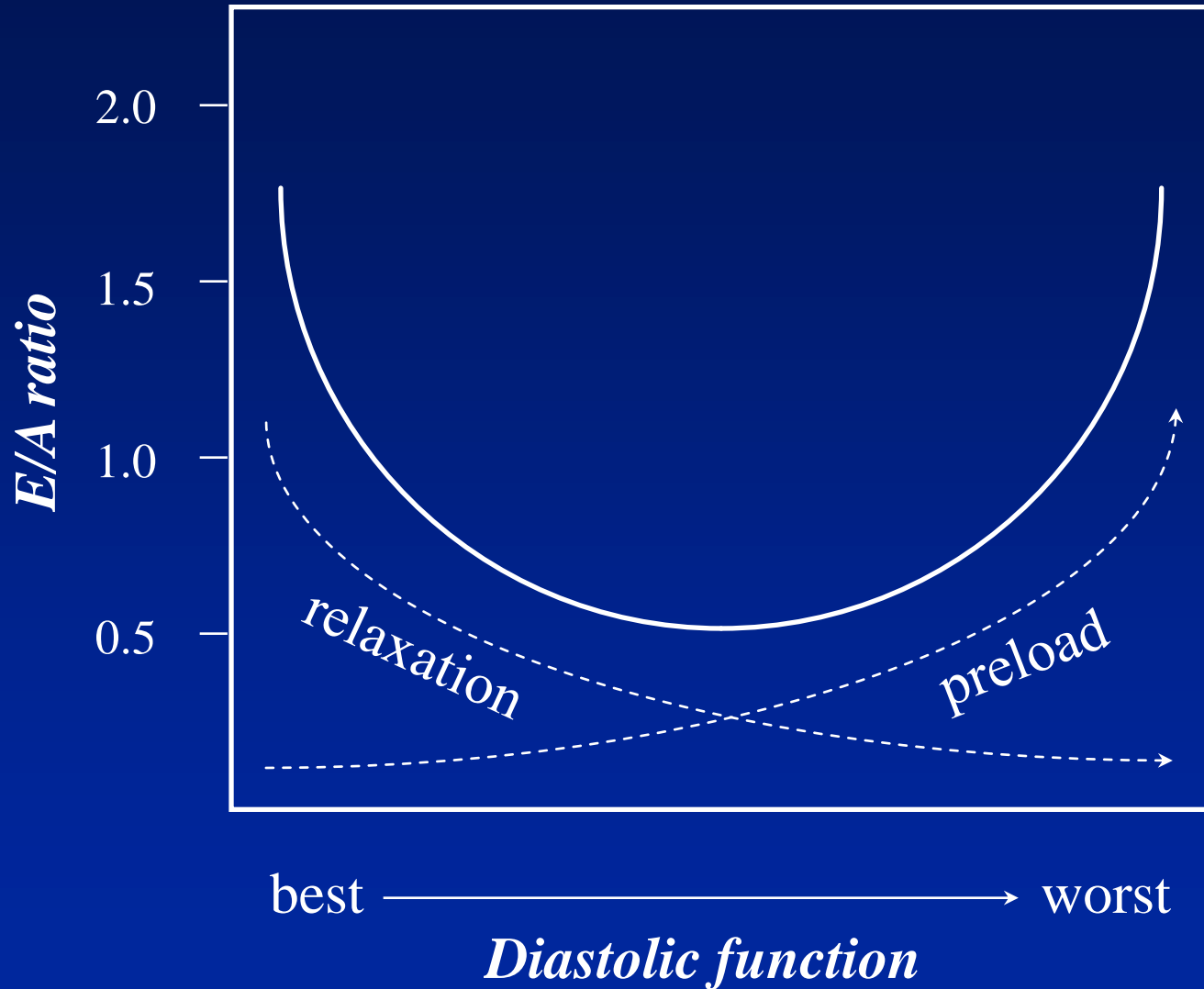
*Pseudonormal*



*Restrictive*



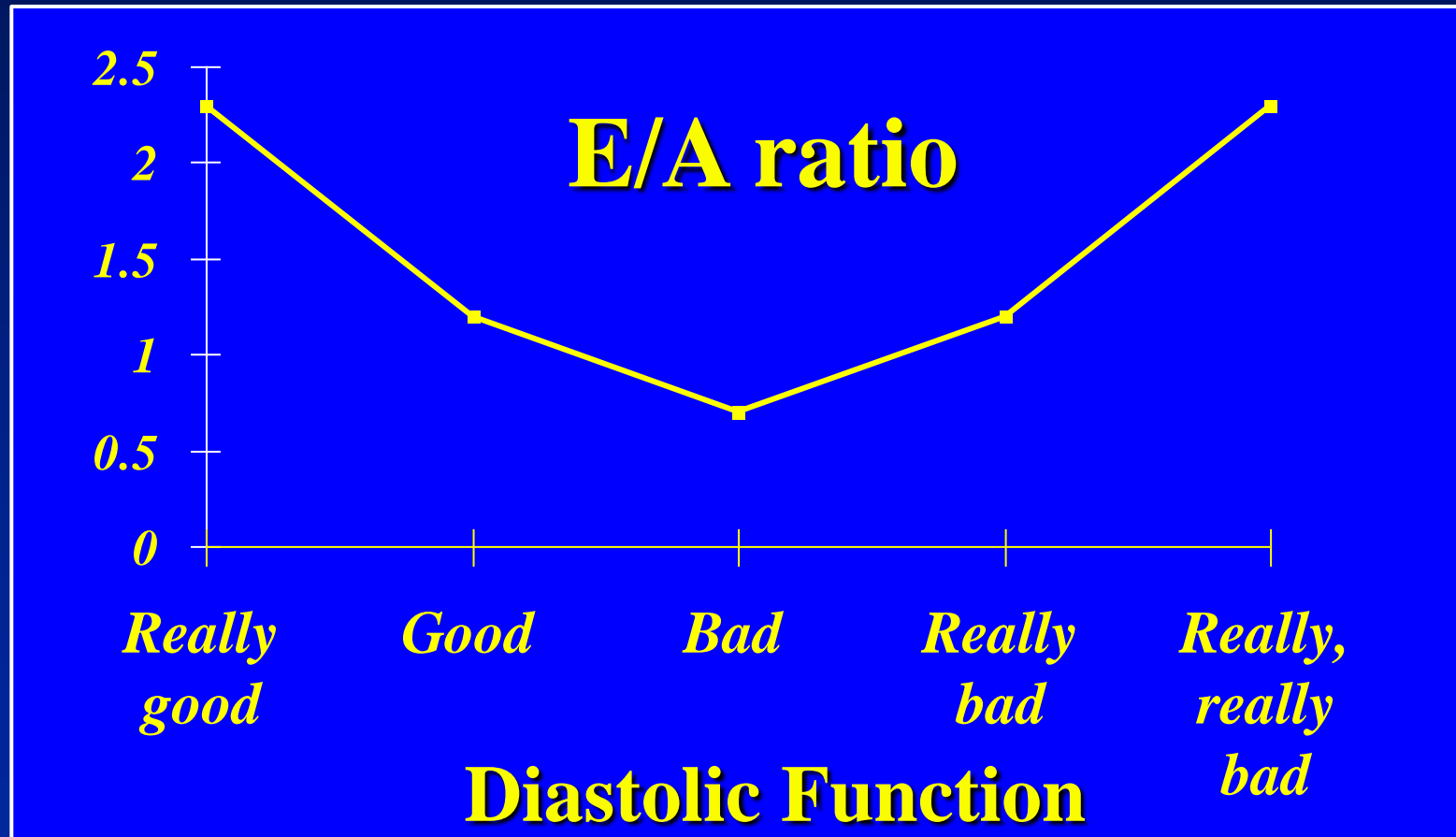
# Preload vs Relaxation *Confounding Effects*



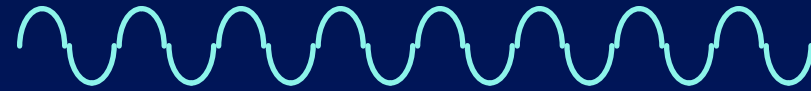
Garcia et al, JACC 1998;32:865



# The Problem with Transmitral Doppler Assessment of Diastolic Function *Preload Sensitivity*



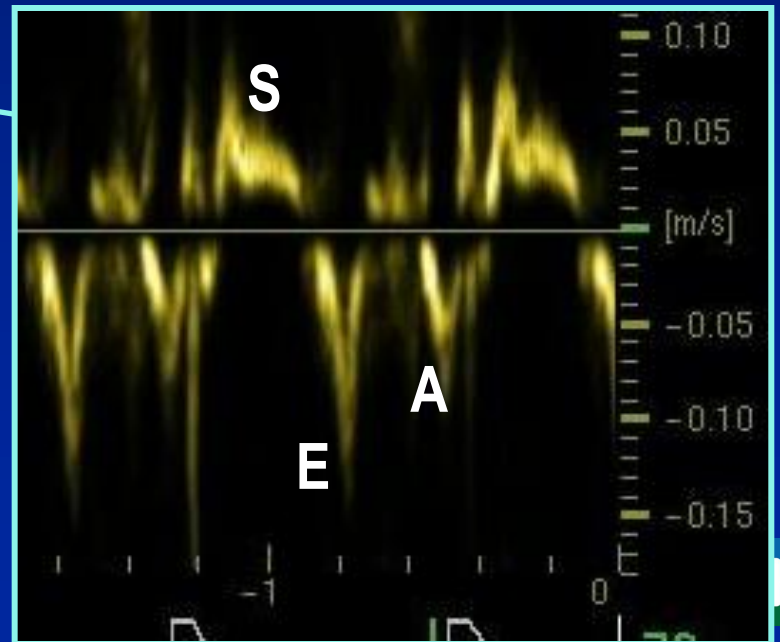
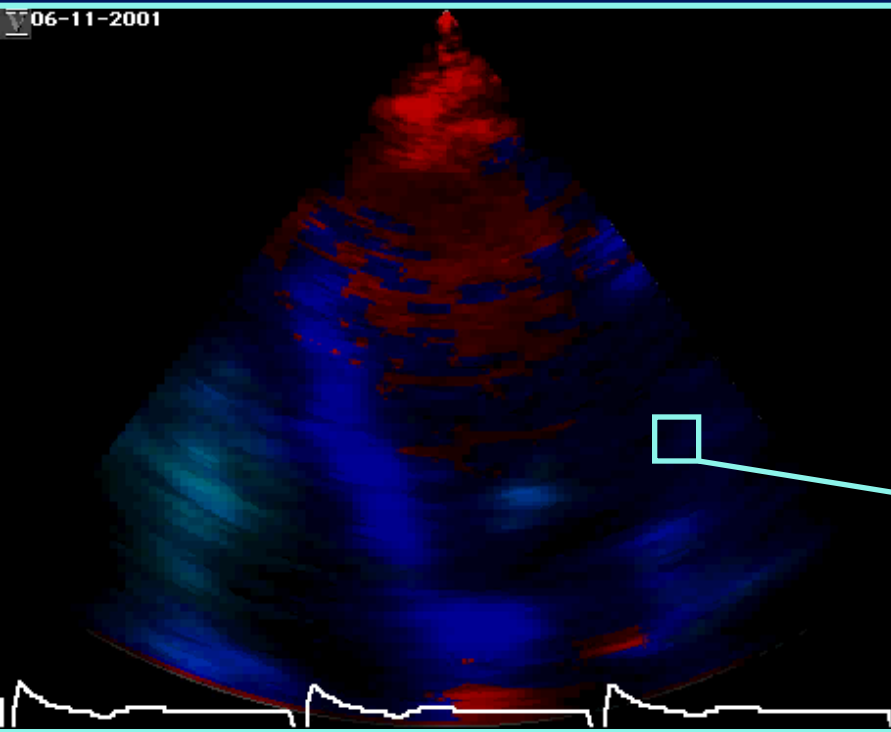
# Tissue Doppler Imaging



*Blood: High velocity, low amplitude*

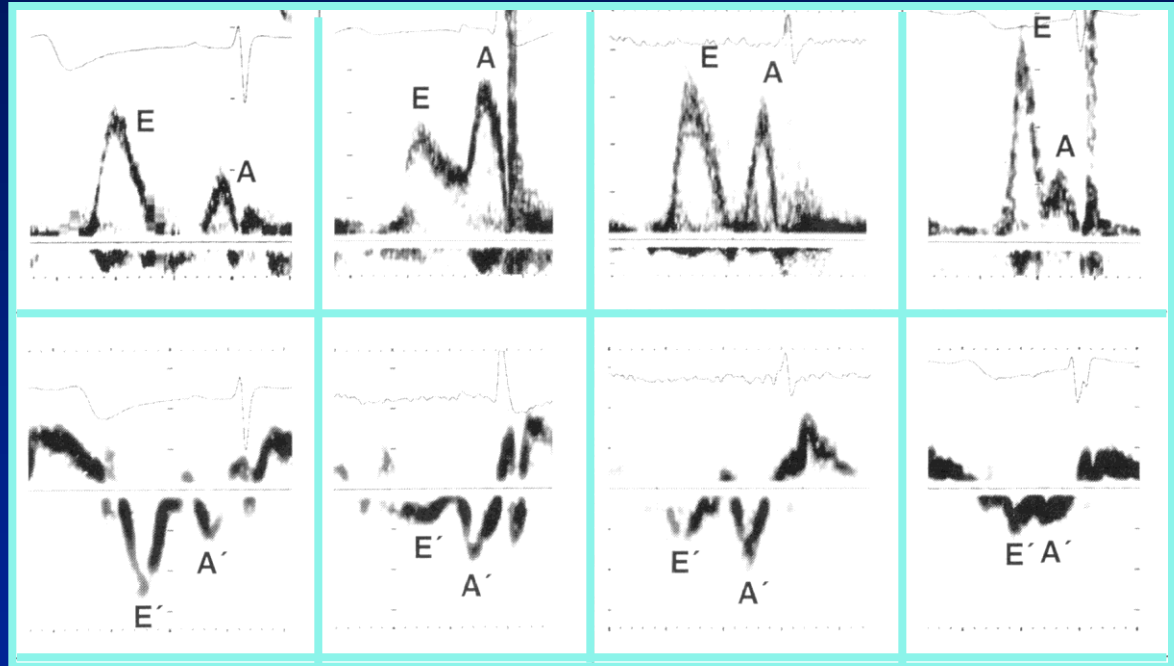


*Tissue: Low velocity, high amplitude*



# Myocardial Wall Velocities *Independence from Mitral Inflow*

*Mitral inflow*



*Mitral annulus*

*Normal*

*Delayed  
relaxation*

*Pseudo-  
normal*

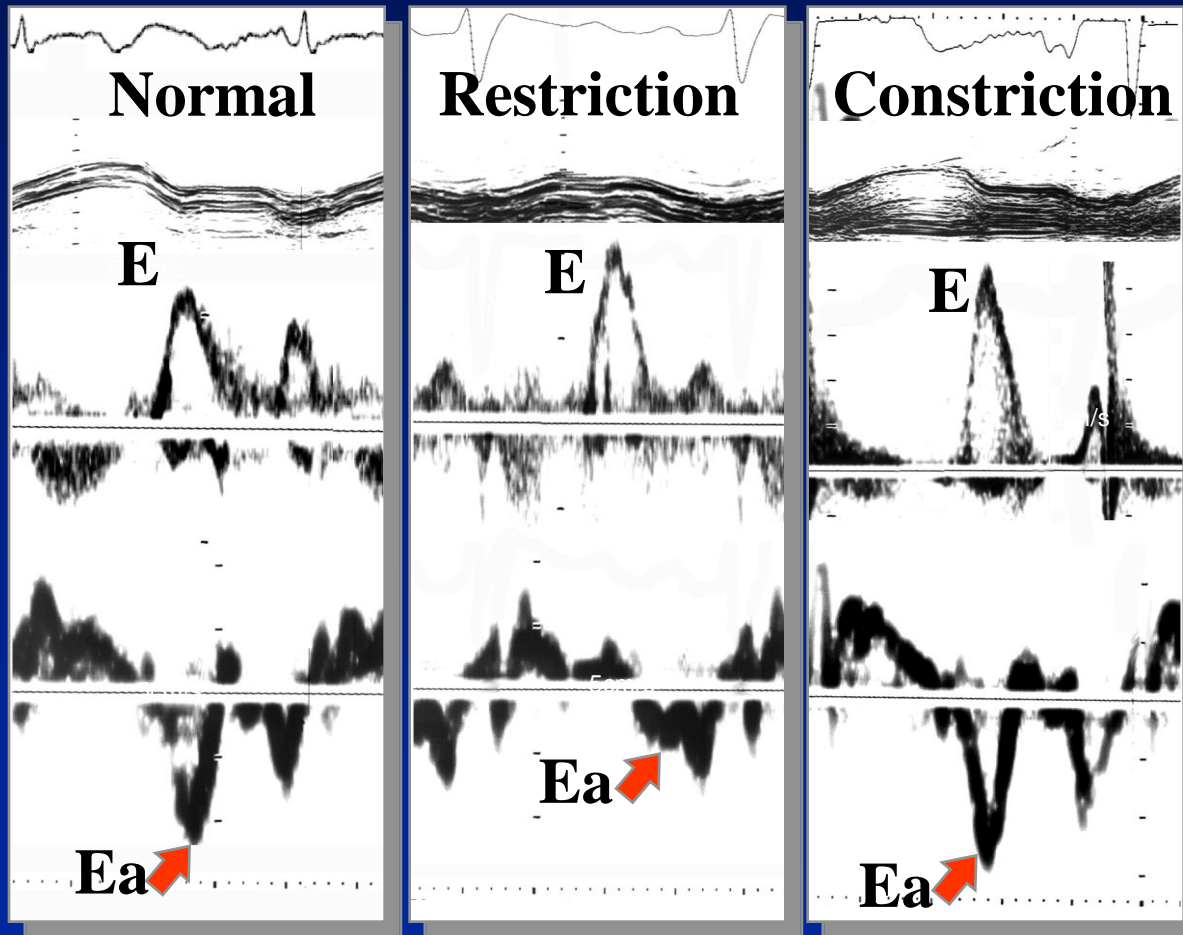
*Restrictive*

# Constriction vs Restriction

*M-mode*

*Doppler*  
*MV Flow*

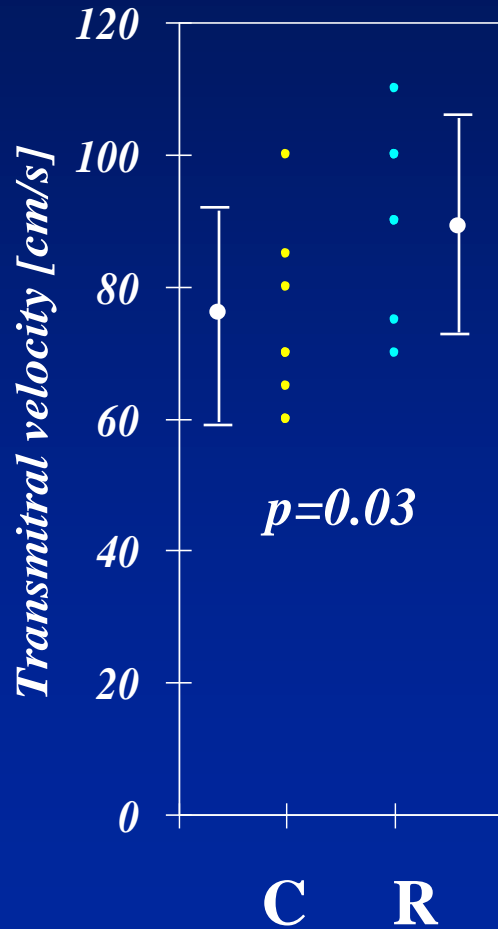
*TDE*



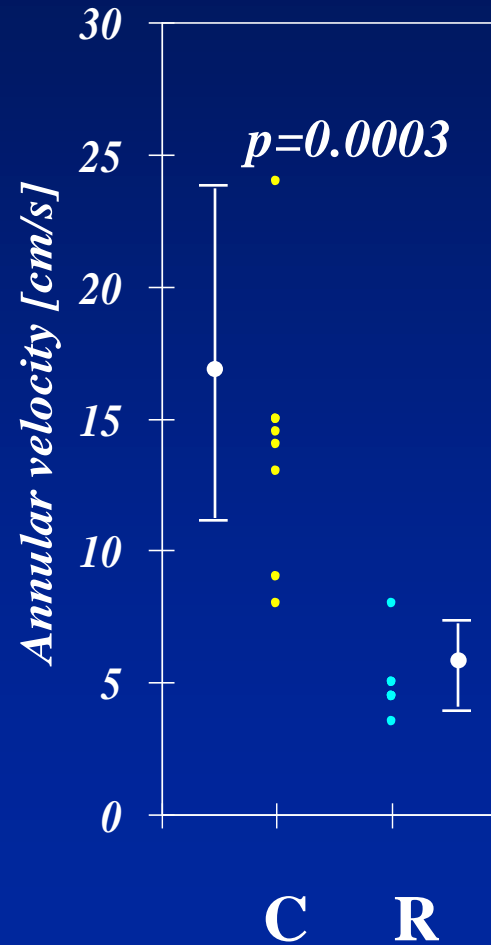
# Constriction vs Restriction

## *Doppler Differentiation*

### *Mitral Pulsed Doppler*

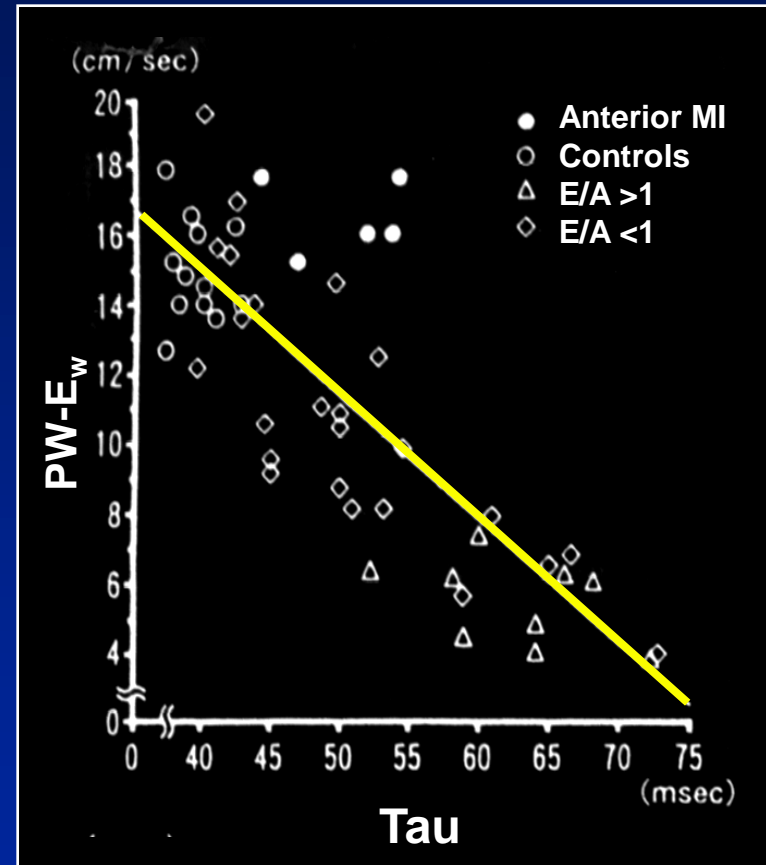
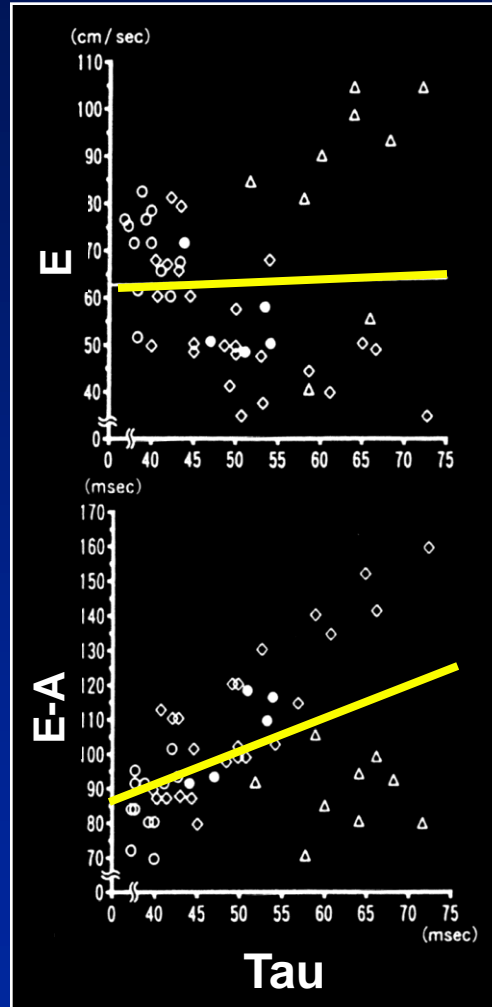


### *Annular DTI*



# Assessment of LV Relaxation

## *DTE E-wave Inversely Related to $\tau$*



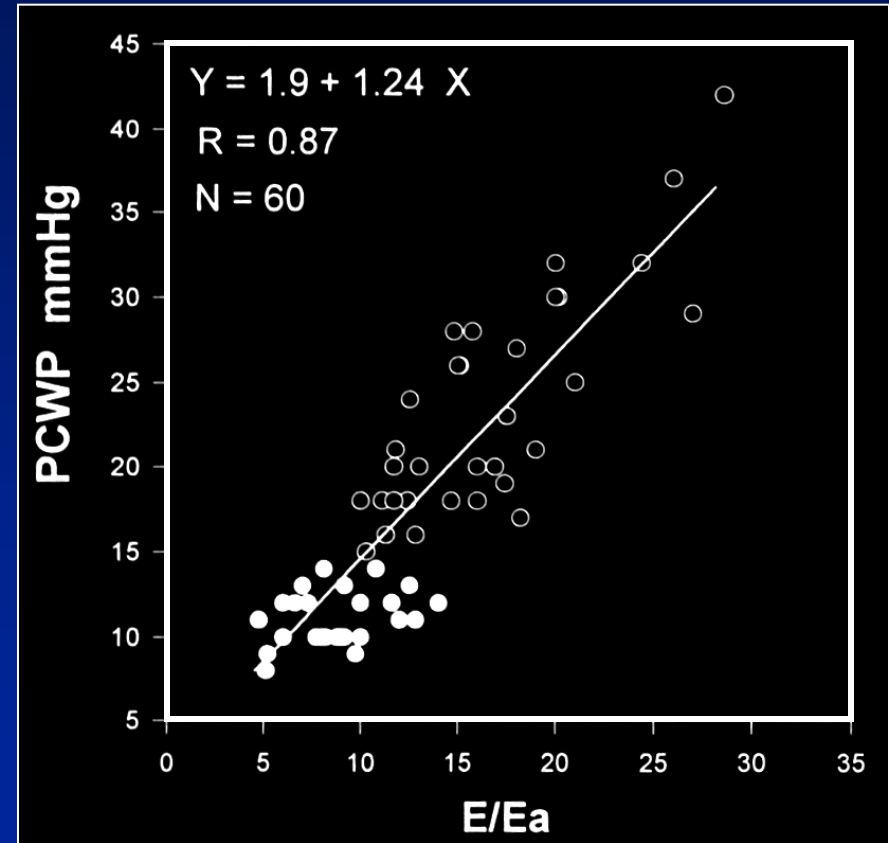
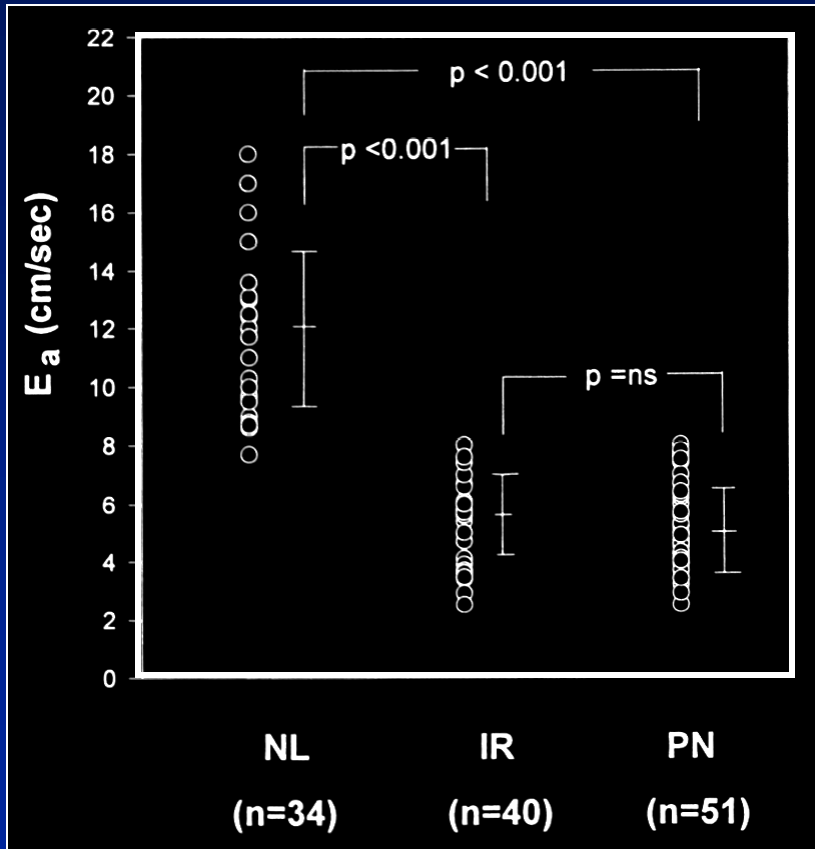
# Implication

*E/Ea Can Predict LA Pressure*

*If  $E \propto LAP/\tau$   
and  $Ea \propto 1/\tau$ ,  
then  $LAP \propto E/Ea$*



# Estimation of $P_{LA}$



*Nagueh et al. JACC 1997;30:1527-33.*



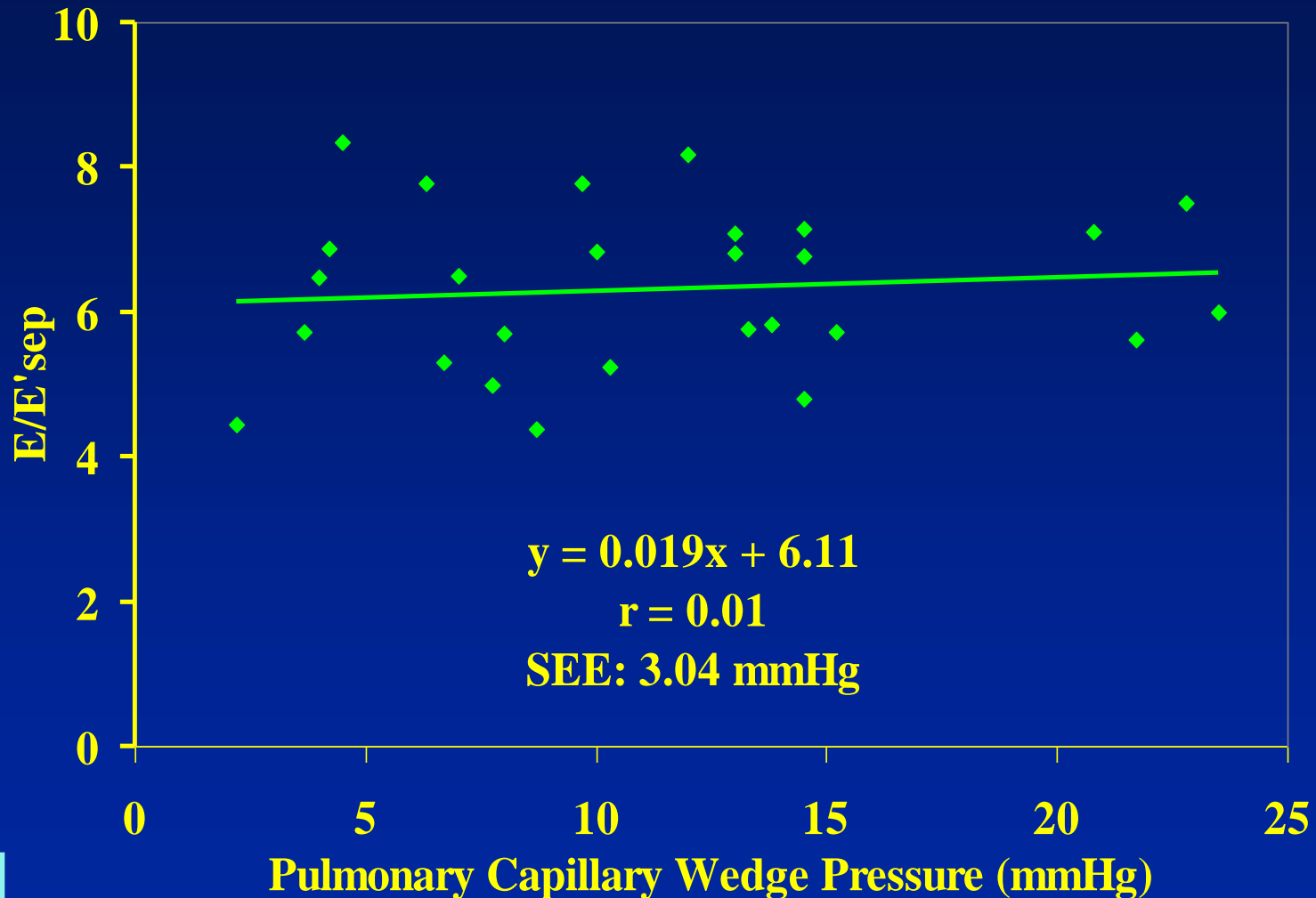
# **Are DTI Velocities Truly Independent of Preload??**

*No, not if the heart is normal*



# Estimation of Left Atrial Pressure

## *Subjects without Heart Disease*

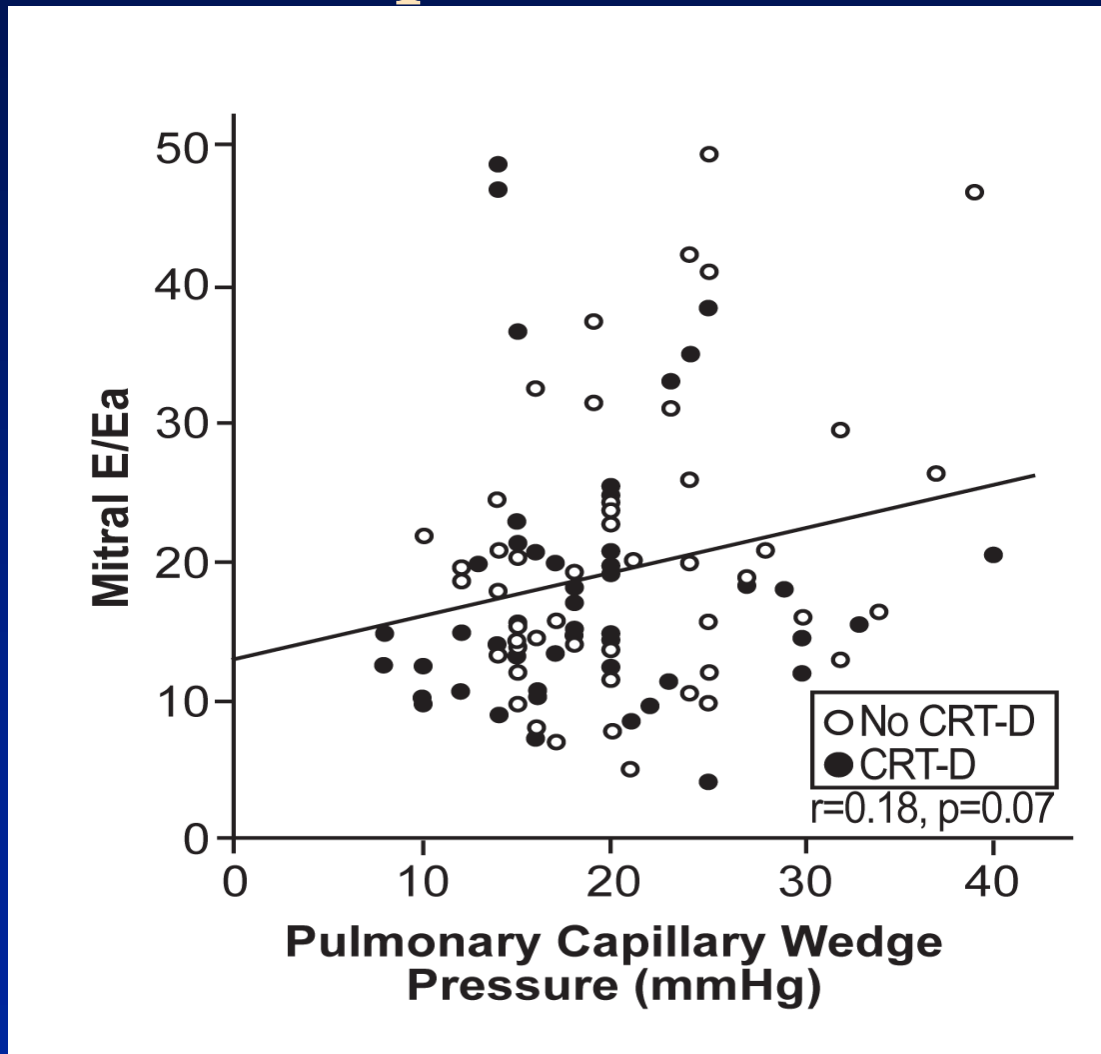


Firstenberg et al. *J Am Coll Cardiol* 2000; 36: 1664-9.



# And Not if the Heart is REALLY Sick

## *Acute Decompensated Heart Failure*

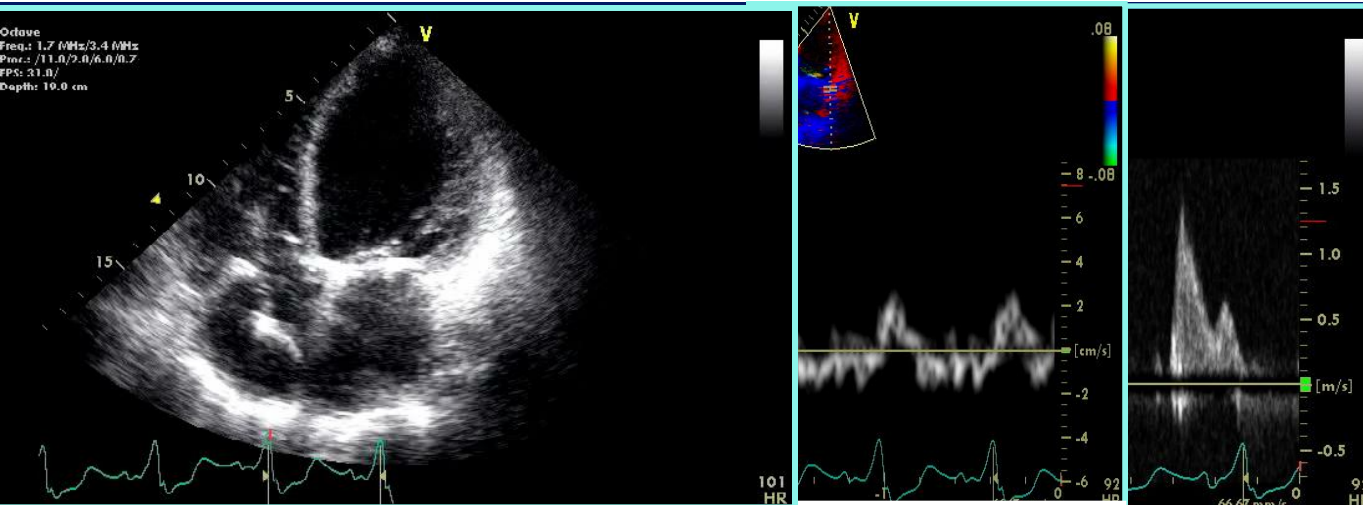


# Tissue Doppler Imaging in the Estimation of Intracardiac Filling Pressure in Decompensated Patients With Advanced Systolic Heart Failure

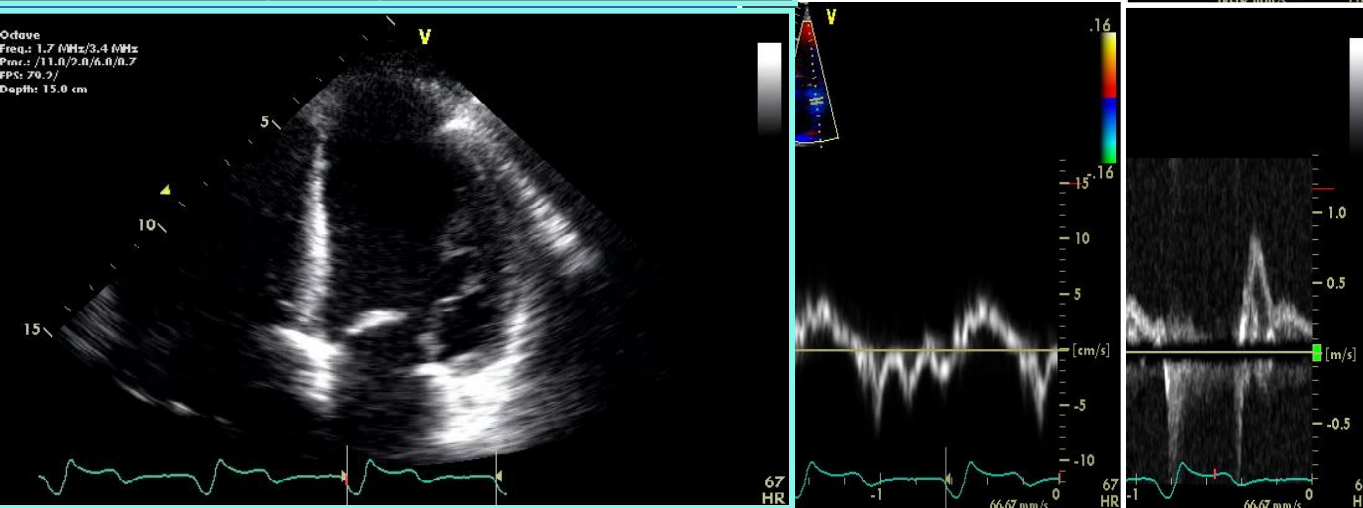
Wilfried Mullens, MD; Allen G. Borowski, RDCS; Ronan J. Curtin, MD;

Circ 2009; 119: 62-70

James D. Thomas, MD; W.H. Tang, MD



**EF = 24%**  
**E = 135 cm/sec**  
**Lateral e' = 2**  
**E/e' = 67**  
**PCW = 14 mmHg**



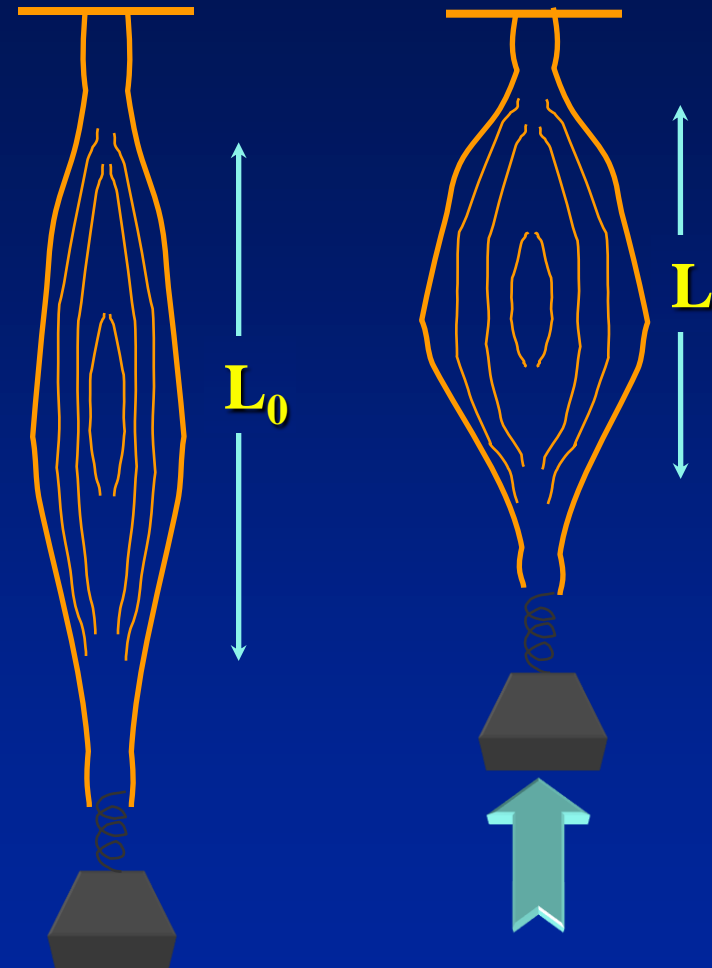
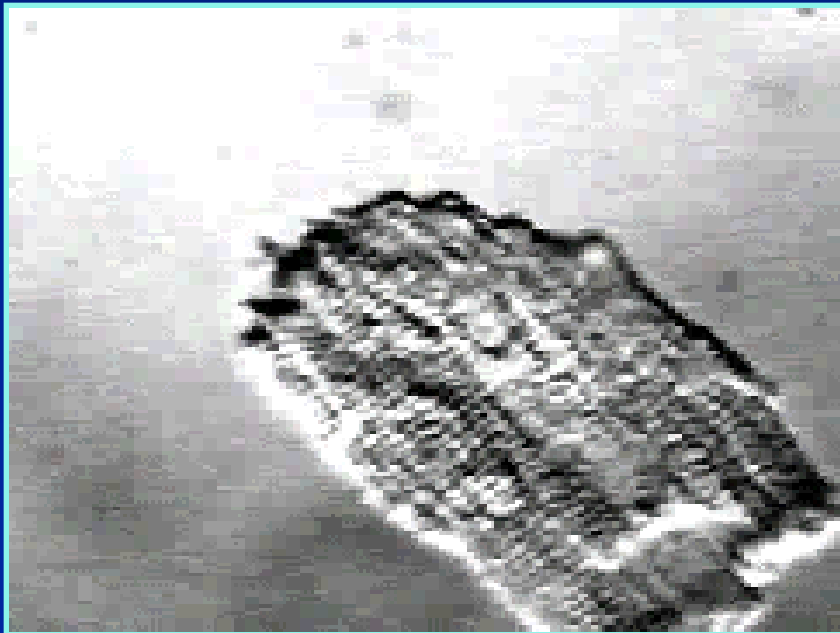
**EF = 31%**  
**E = 89 cm/sec**  
**Lateral e' = 6.9**  
**E/e' = 14**  
**PCW = 33 mmHg**

# Myocardial Strain: What is It??

**Strain: dimensionless index of change in length**

$$\text{Strain } (\epsilon) = \frac{L - L_0}{L_0}$$

**LV strain may offer a pure index of regional LV function but is difficult to measure**



# Predictors of Systolic and Diastolic Strain Rate

*Relationship to invasive indices during ischemia*

## Systolic Strain Rates

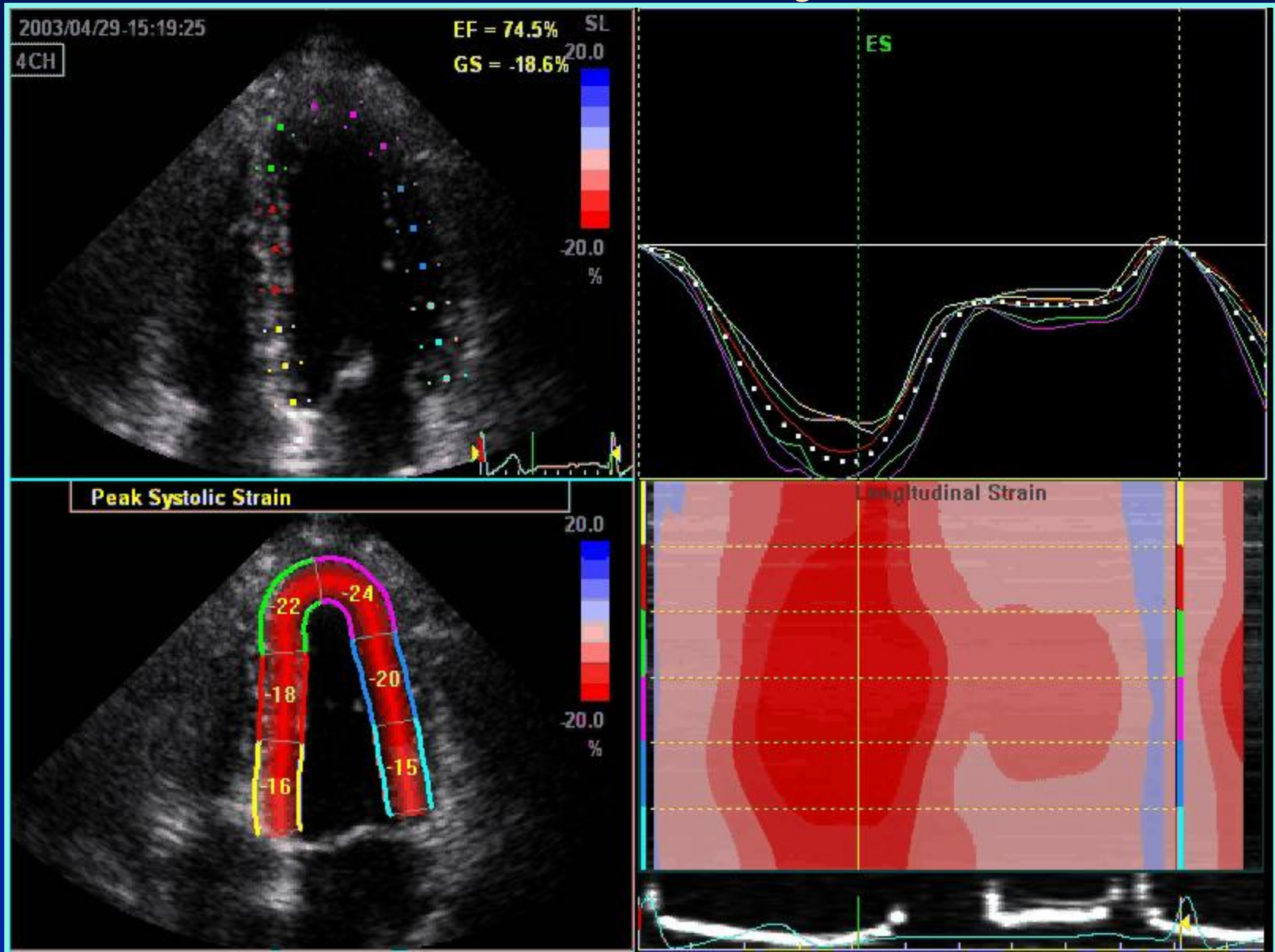
$\epsilon'_{\text{SYS}}$ VS:	r
<b>ES P/V</b>	<b>0.89</b>
<b>+dP/dt<sub>max</sub></b>	<b>0.86</b>
<b>EF</b>	<b>0.77</b>
<b>ESV</b>	<b>0.57</b>

## Diastolic Strain Rates

$\epsilon'_{\text{DIAS}}$ VS:	r
<b>EDP</b>	<b>0.82</b>
<b>-dP/dt<sub>max</sub></b>	<b>0.81</b>
<b>Tau</b>	<b>0.72</b>
<b>EDV</b>	<b>0.60</b>

# Longitudinal Strain from B-Mode Echo

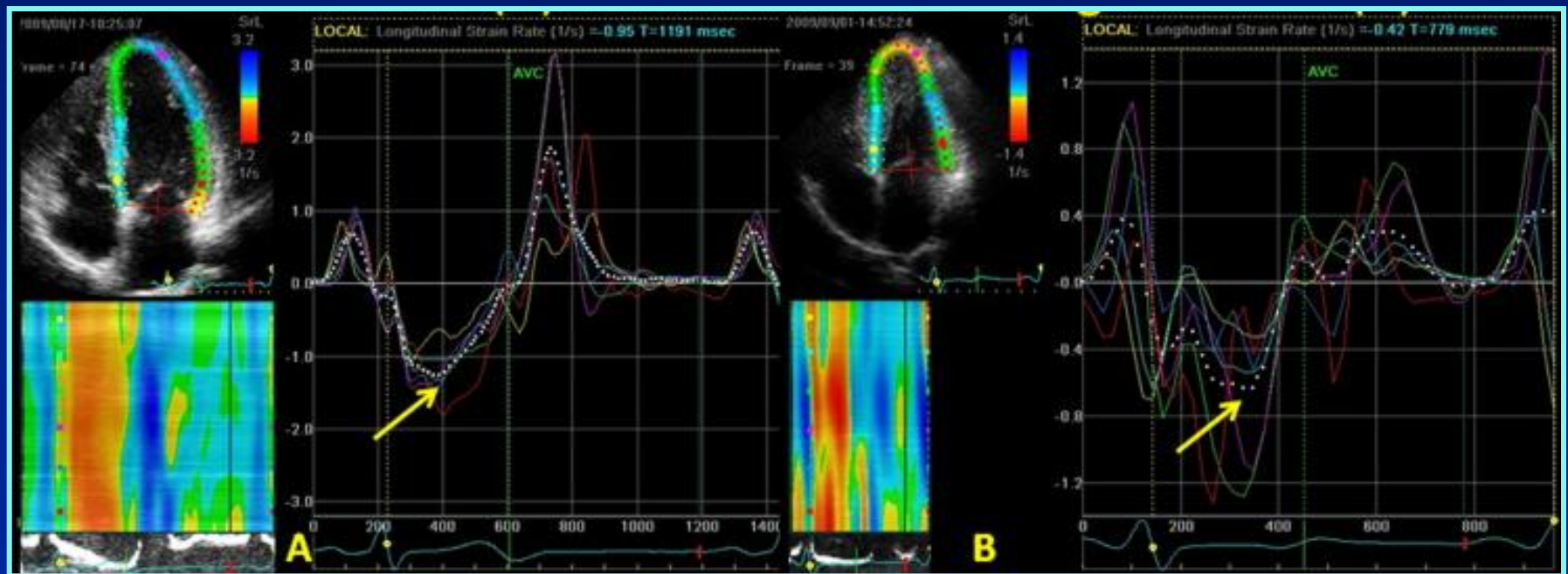
## *Normal Subject*



# Systolic and Diastolic Myocardial Mechanics in Patients with Cardiac Disease and Preserved Ejection Fraction: Impact of Left Ventricular Filling Pressure

John S. Nguyen, MD, Nasser M. Lakkis, MD, Jaromir Bobek, RCIS, Rajiv Goswami, MD, and Hisham Dokainish, MD, *Dallas, Texas; Hamilton, Ontario, Canada*

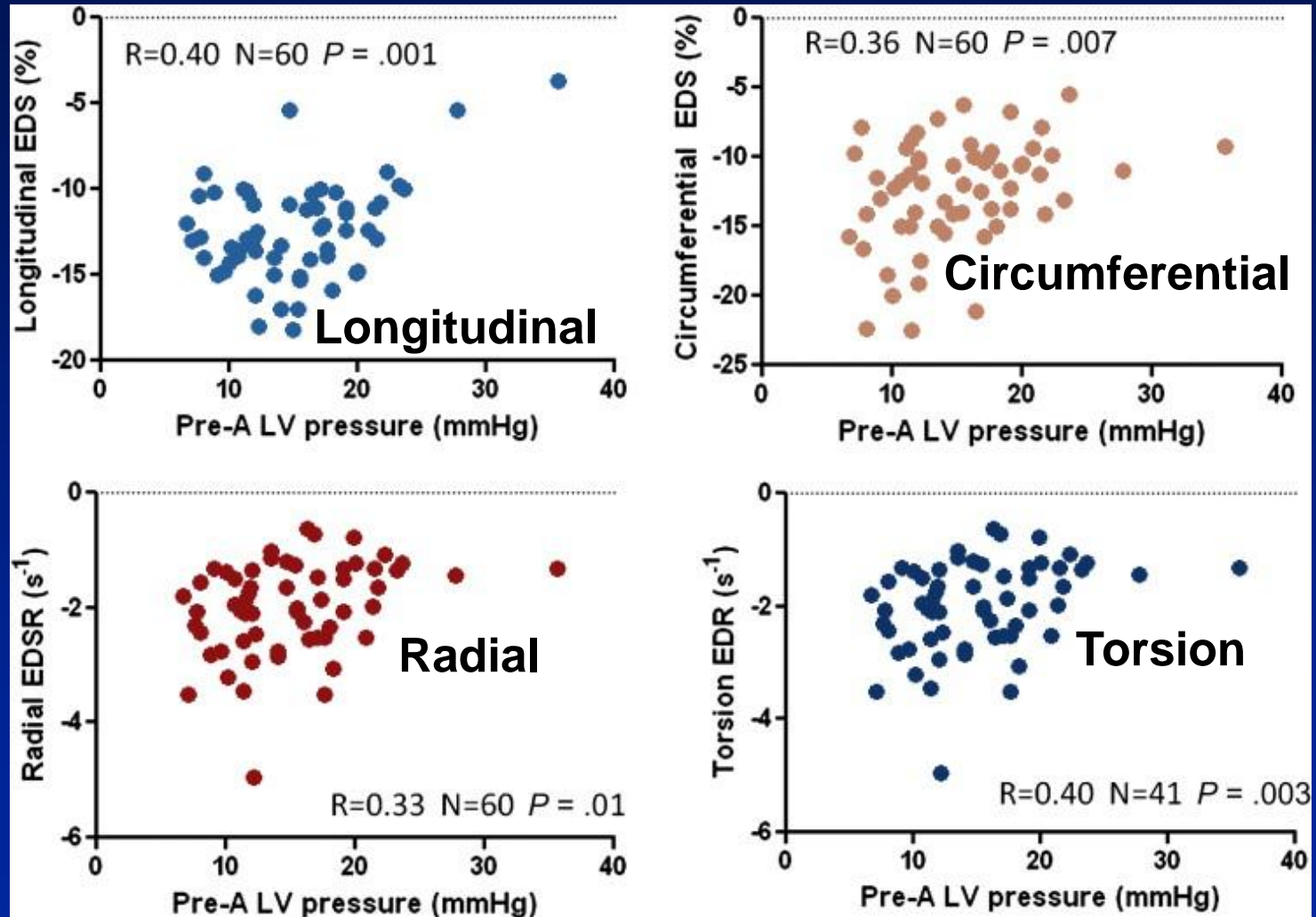
60 pts w/ EF > 50%, 30 w/ pre-A LVEDP < 15 mmHg, 30 over 15 mmHg  
**Normal EDP** **Elevated EDP**



*Higher LVEDP ⇒ lower systolic and diastolic strain rate*

# Diastolic Strain vs LVEDP

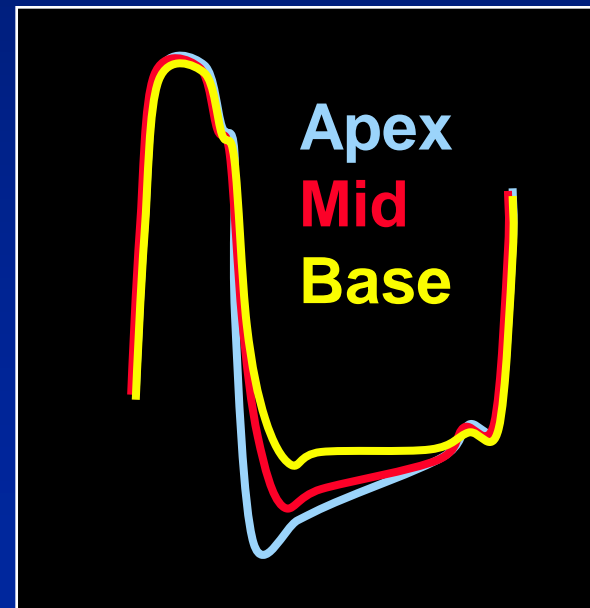
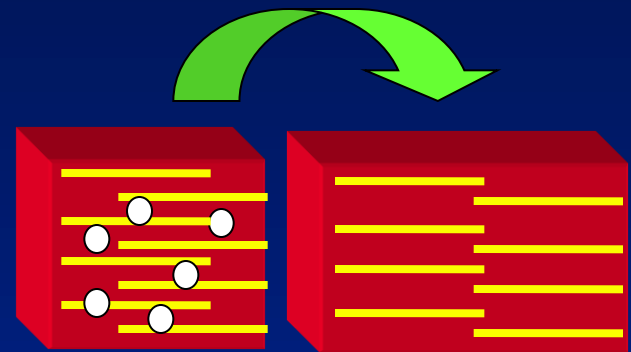
*Lower values  $\Rightarrow$  higher EDP*



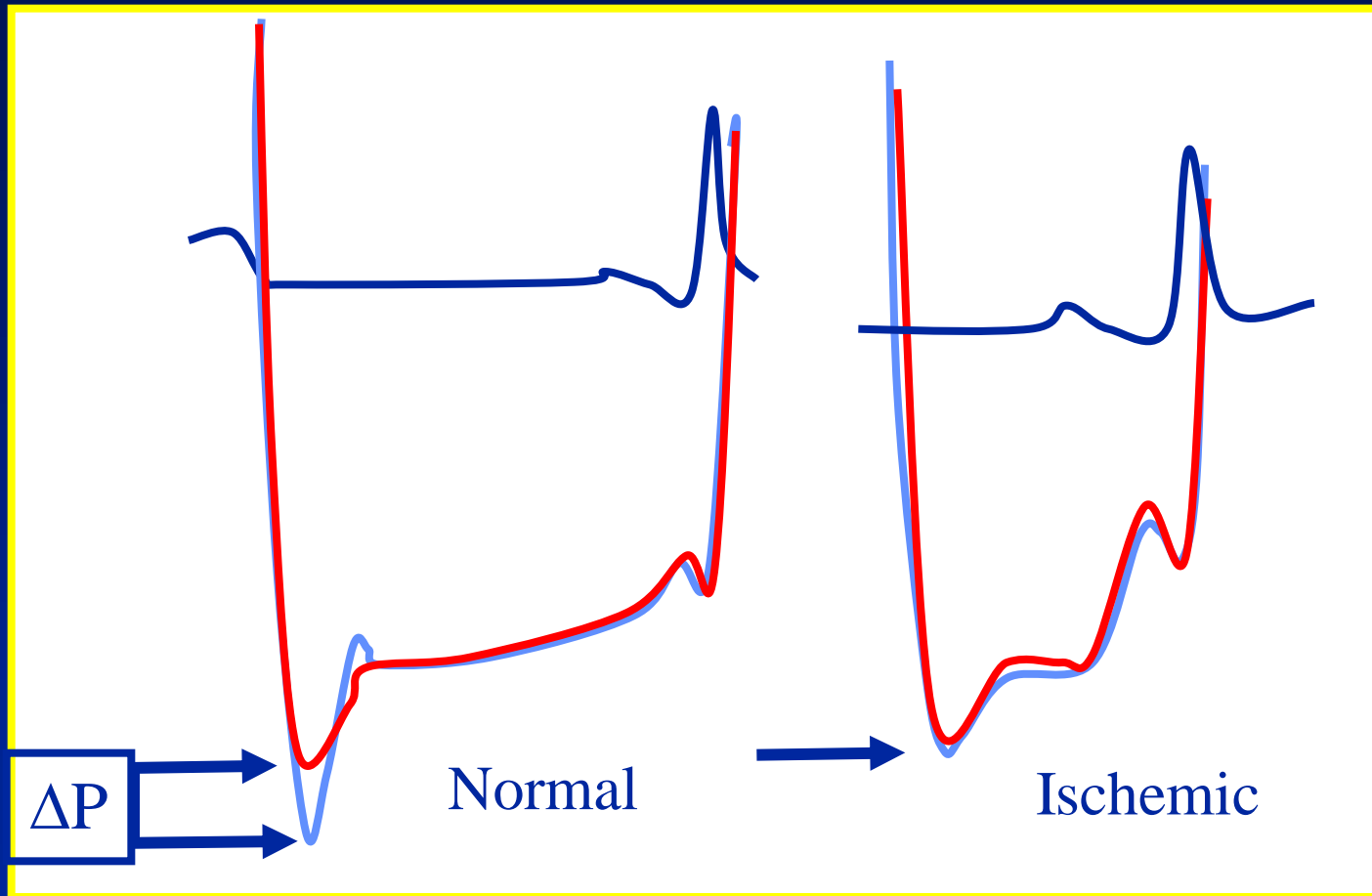
*But  $r^2$  only 0.11 to 0.16*

# LV Relaxation and Intraventricular Pressure Gradients

- Active relaxation, occurring earlier and more rapidly at the LV apex generates small (1-2 mmHg) **intraventricular pressure gradients** in the normal heart.
- These **intraventricular pressure gradients** assist in the low pressure filling of the heart
- Critically impacted by delayed relaxation and dyssynchrony

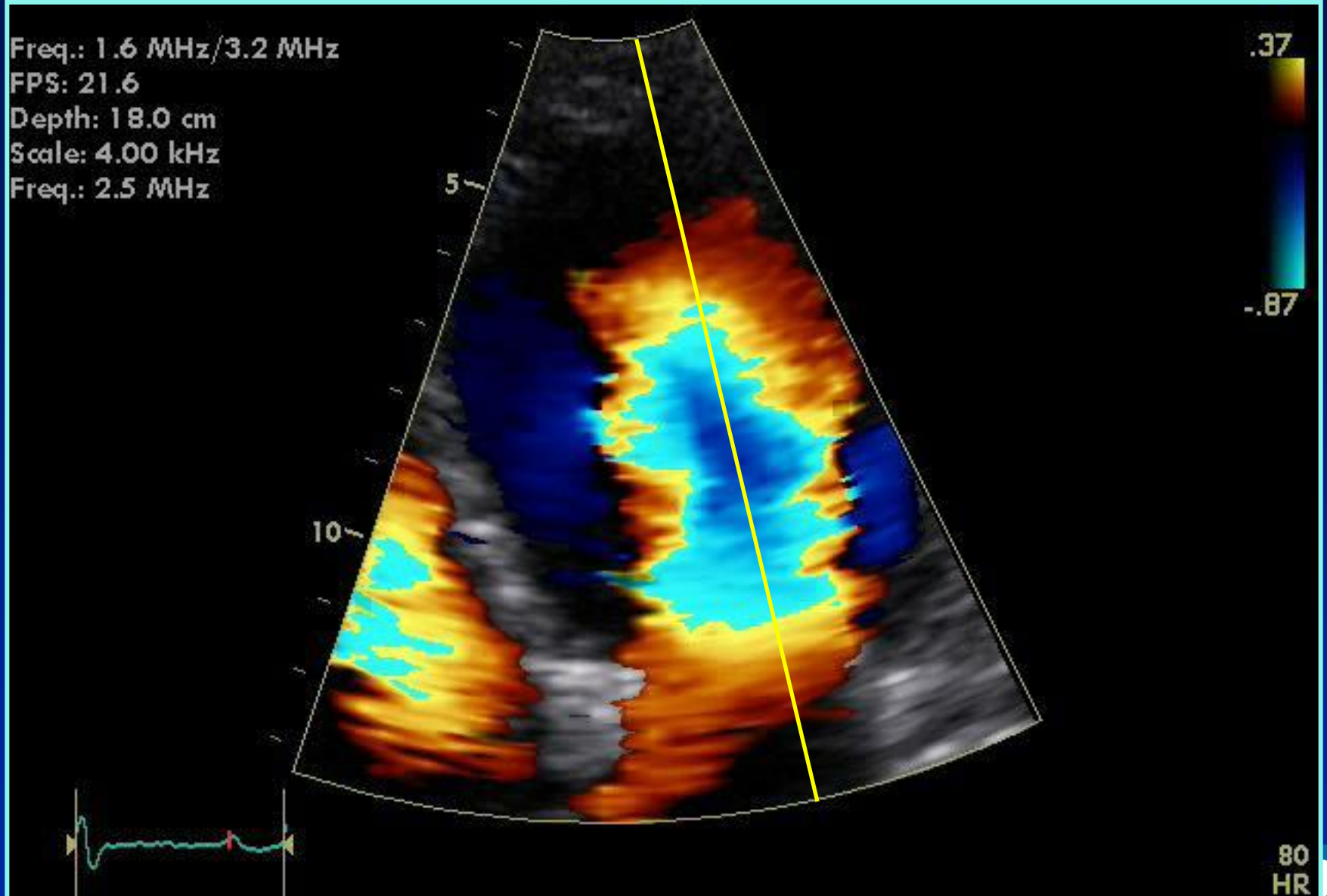


# Importance of Diastolic Suction

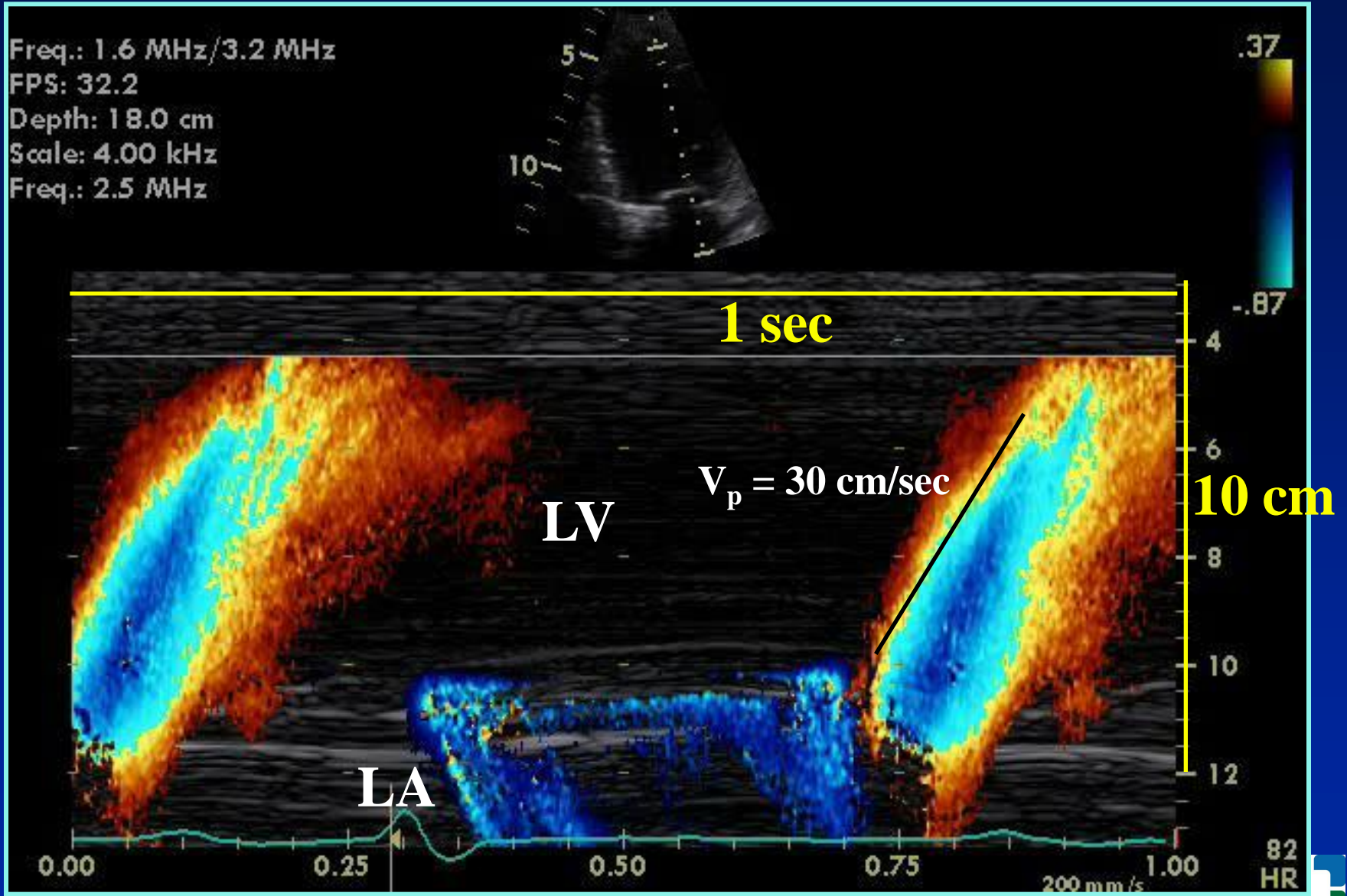


*Almost impossible to measure invasively.....*

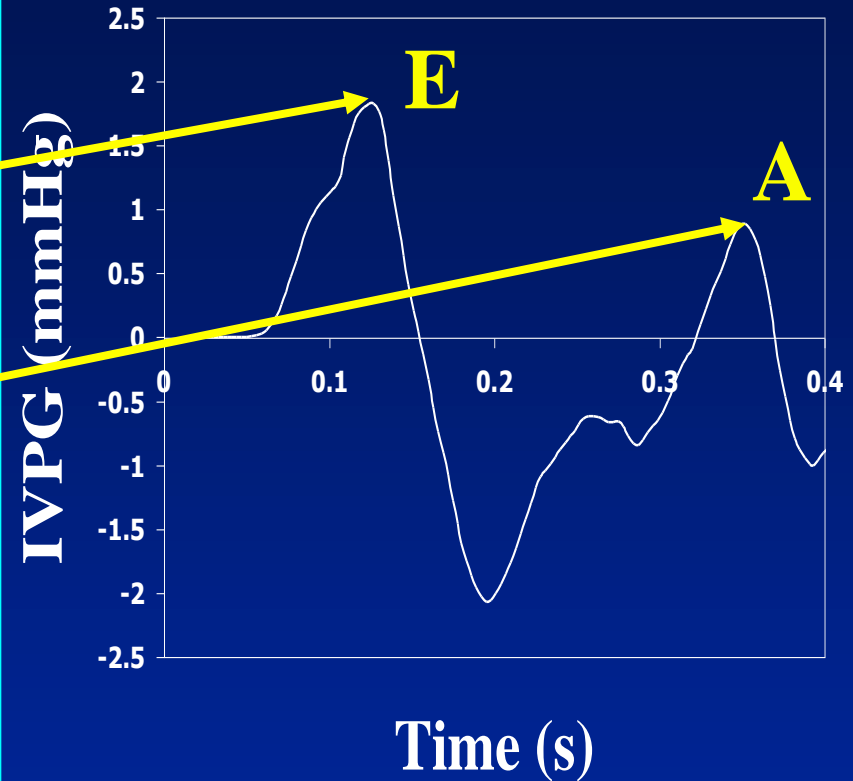
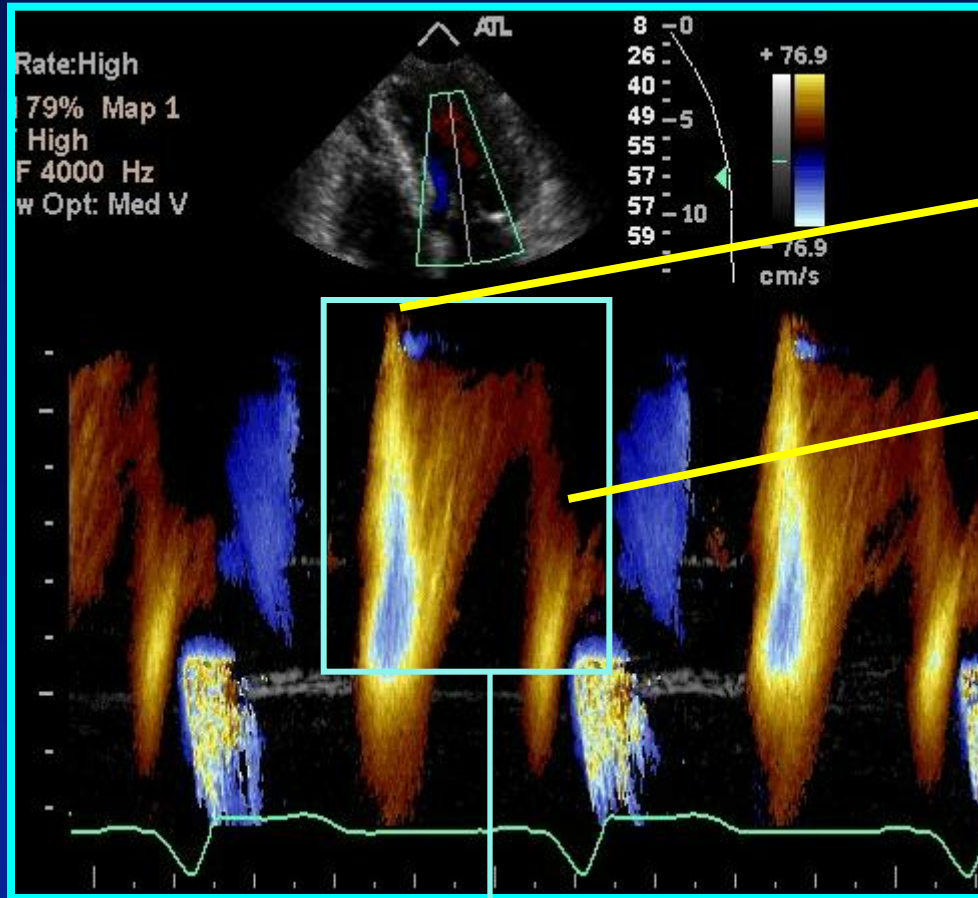
# Color M-Mode Methodology



# Measurement of Propagation Velocity



# CMM Calculation of IVPG

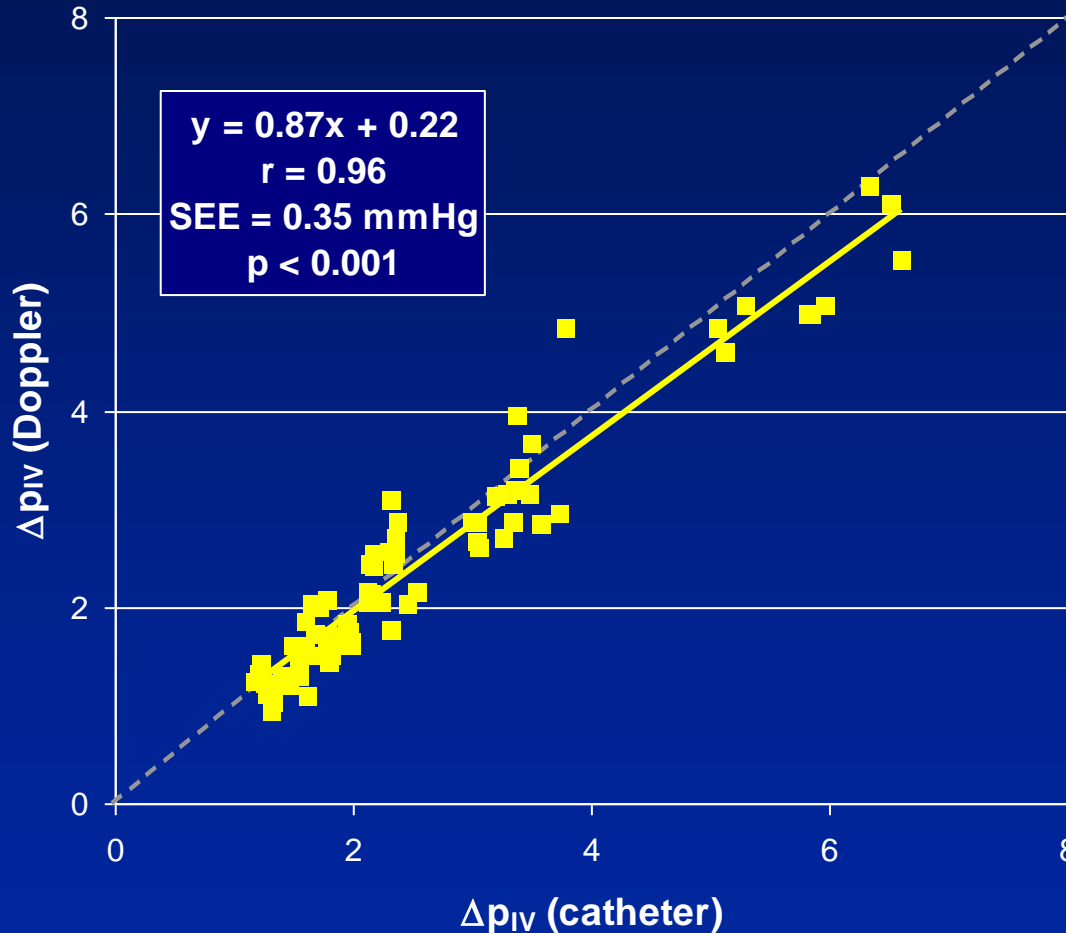


$$\Delta P = -\rho \int_{LV\text{base}}^{LV\text{apex}} \left( \frac{\partial v}{\partial t} + v \frac{\partial v}{\partial s} \right) ds$$

**Euler equation**

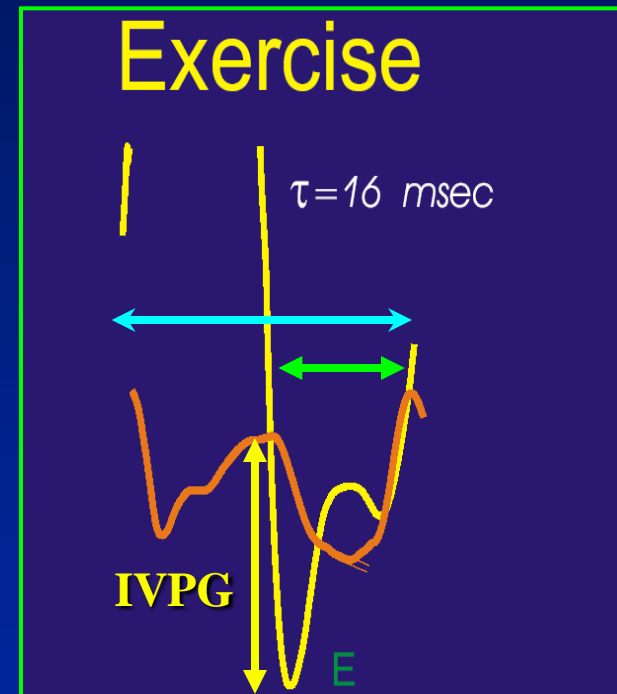
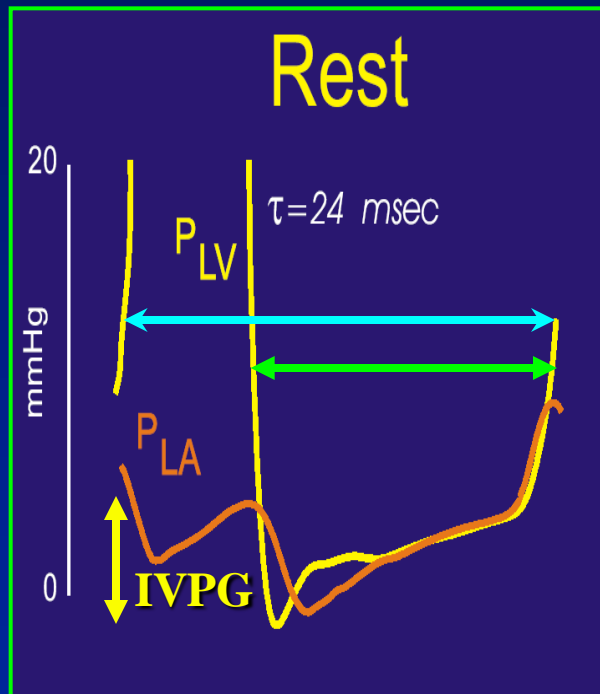


# Intraventricular Pressure Gradient



# IVPG are Critical During Exercise

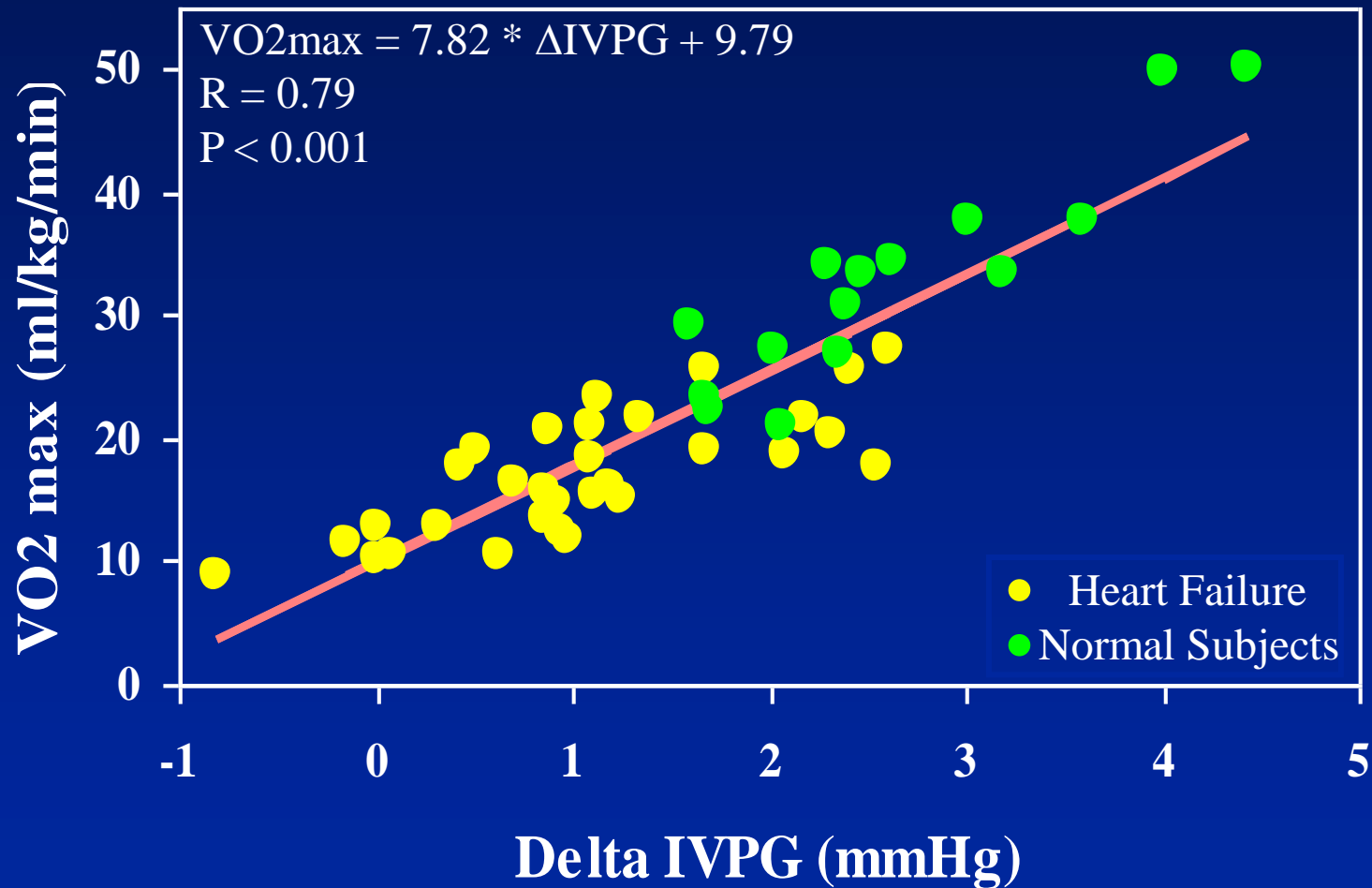
## *Diastole Disproportionately Shortened*



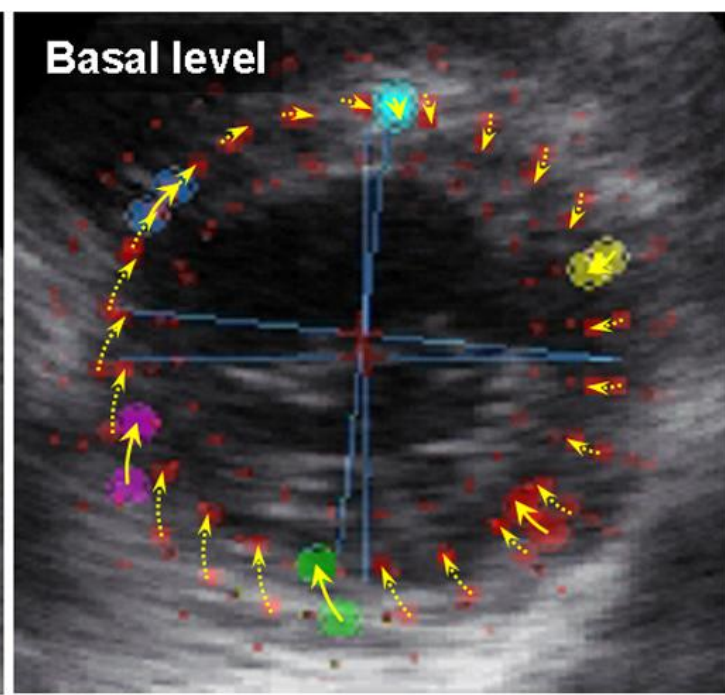
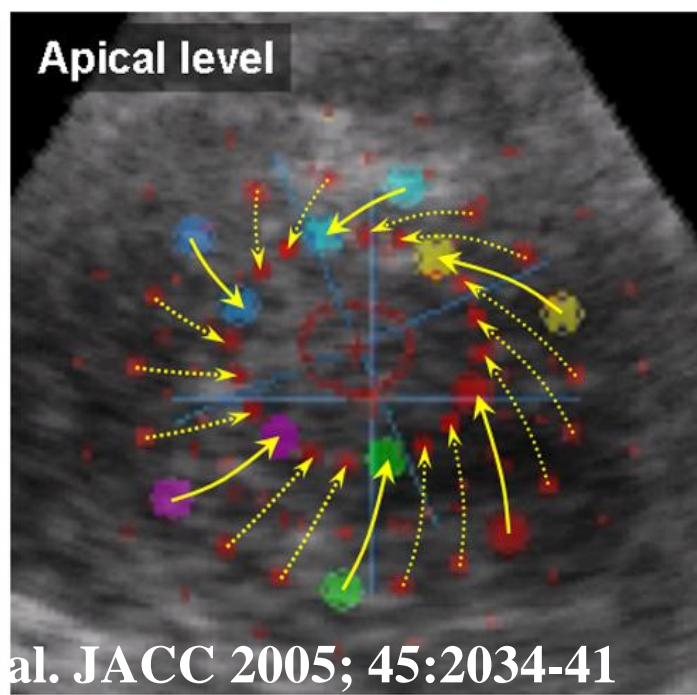
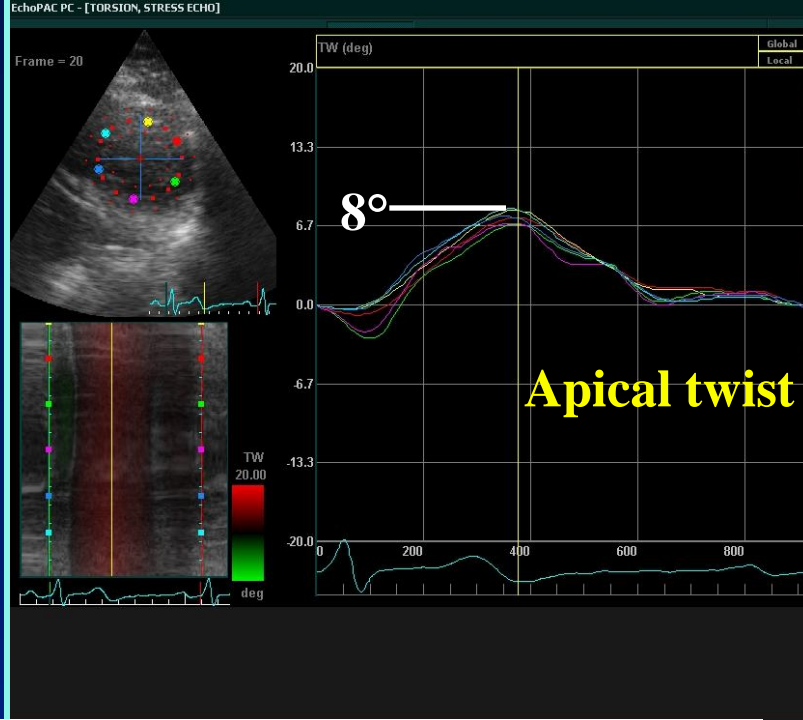
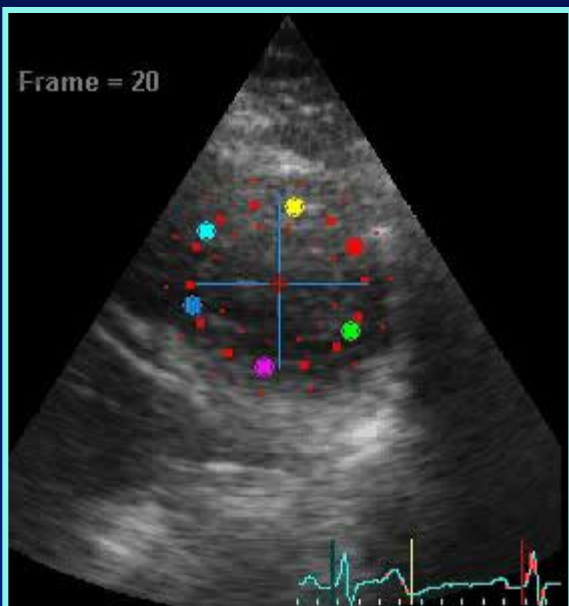
↔ Diastolic Filling Time

↔ Full Cardiac Cycle

# Augmentation of Diastolic Suction Predicts Exercise Capacity

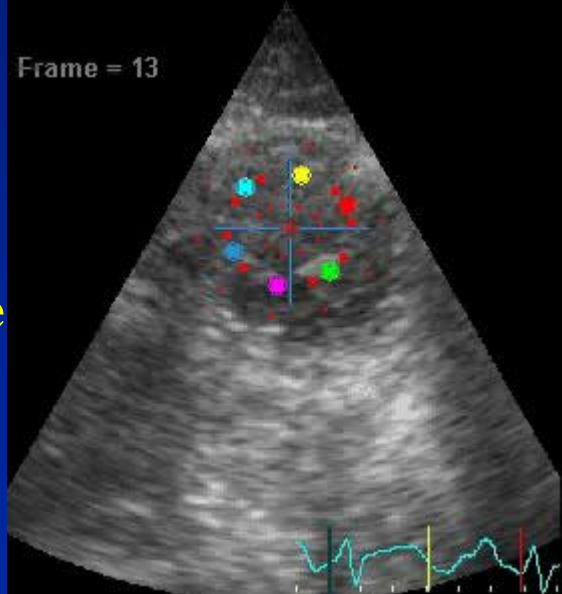
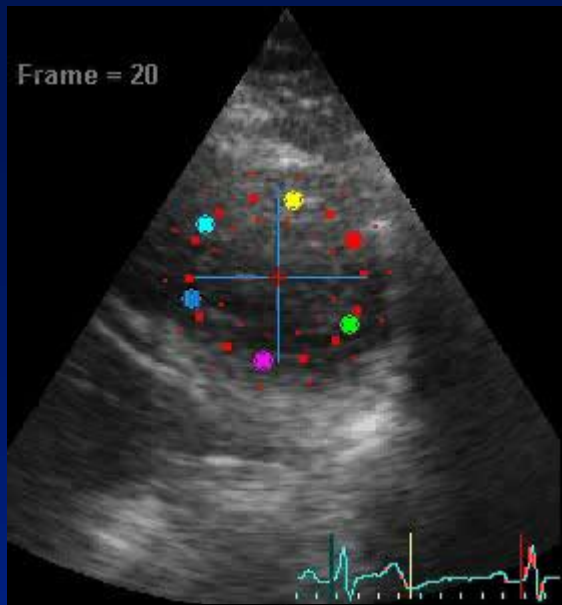


# Torsion from 2D Echo

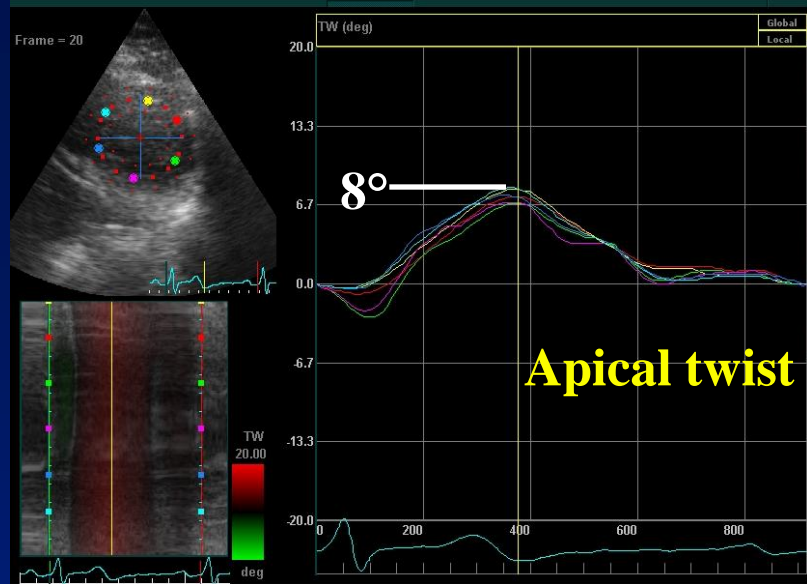


# Torsion During Exercise

Rest

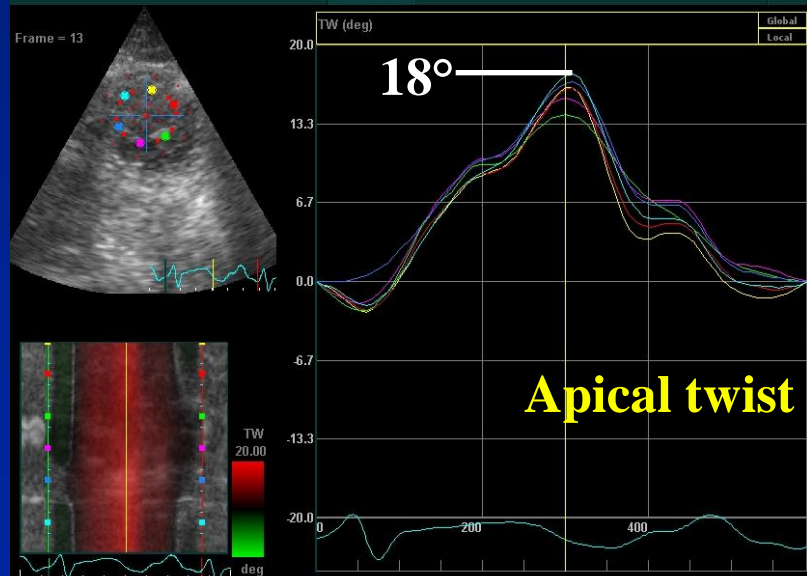


EchoPAC PC - [TORSION, STRESS ECHO]



Apical twist

EchoPAC PC - [TORSION, STRESS ECHO]



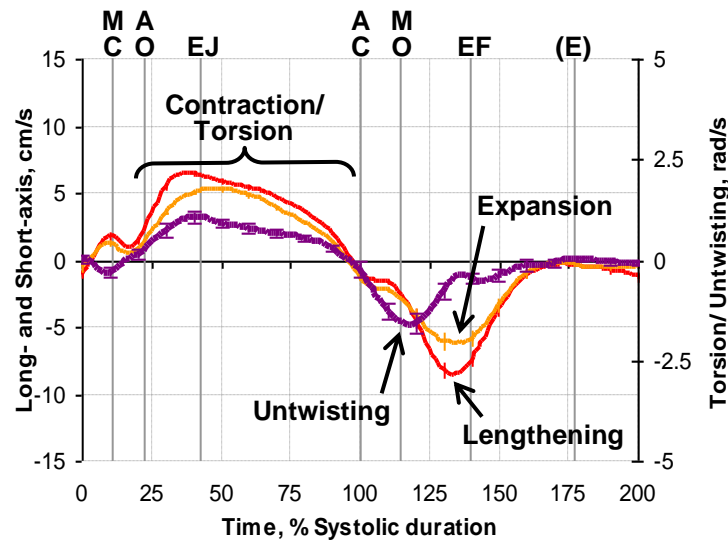
Apical twist



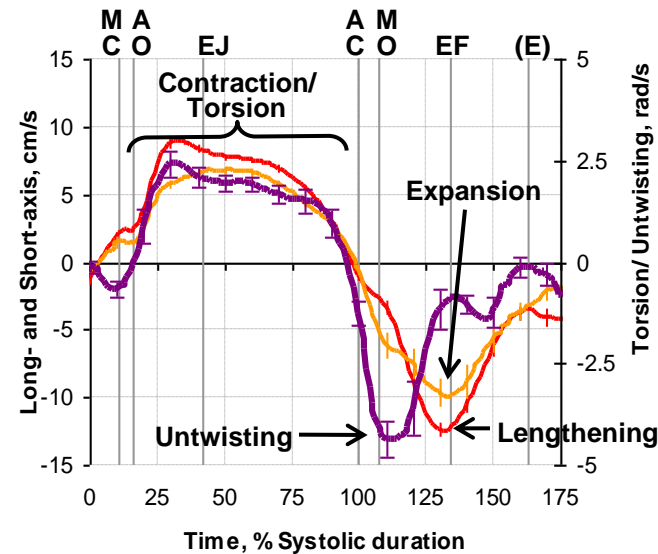
# Timing and Magnitude of LV Mechanics

## *Untwisting is the First Event of Diastole*

Rest

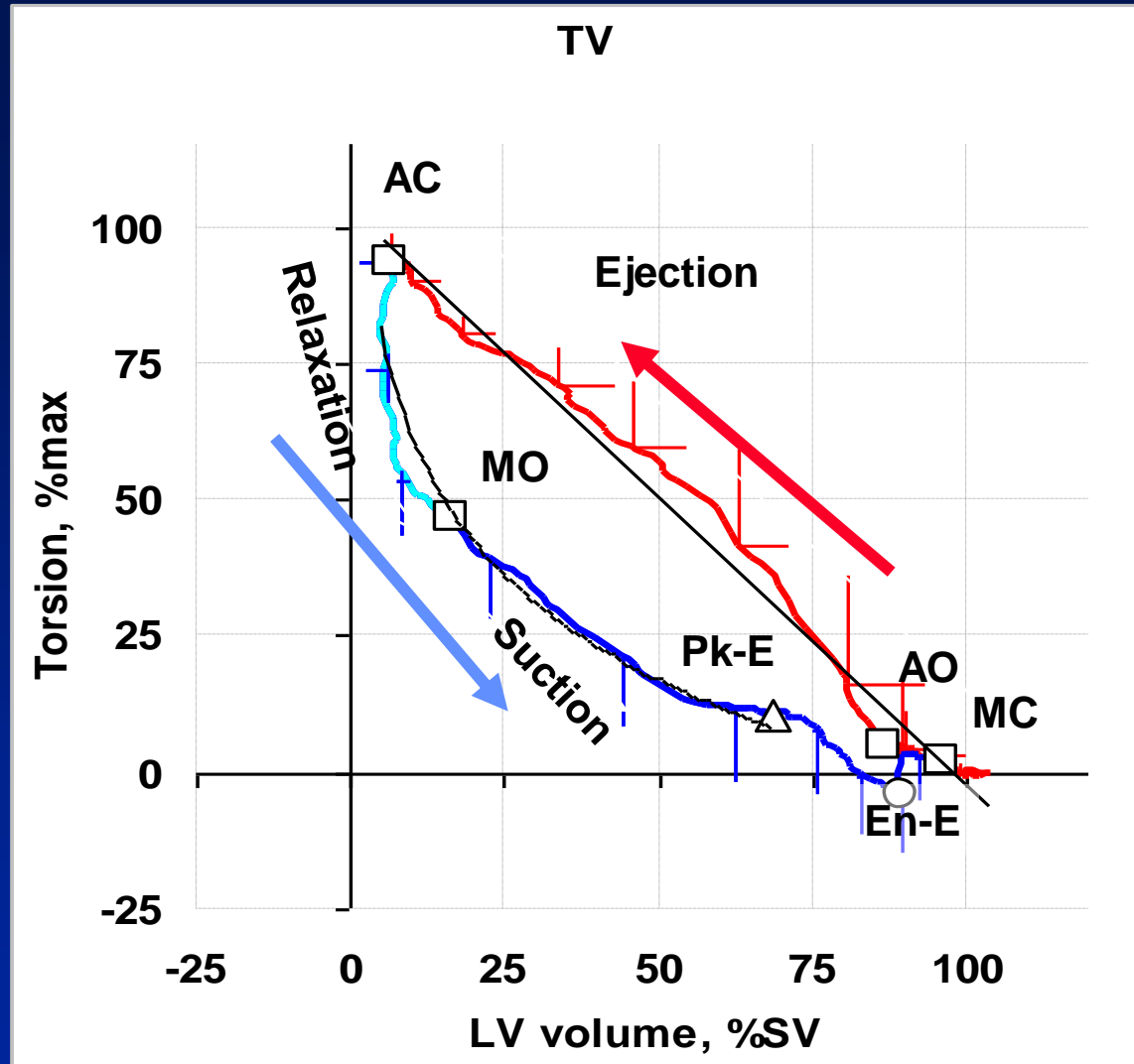


Exercise



- Long axis motion
- Radial motion
- Torsion

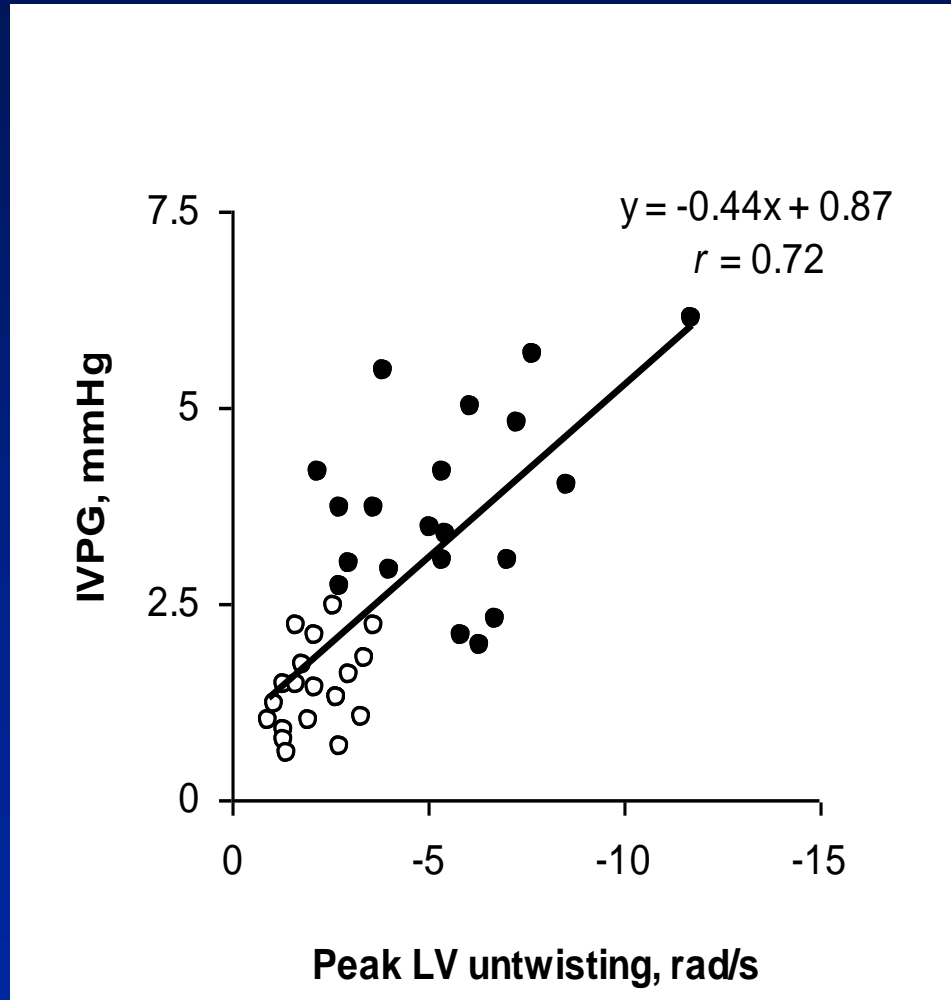
# Torsion-Volume Loop



*Half of torsion is released before MV opens*



# LV Untwisting Predicts IVPG



# Putting It All Together

- *During systole, a significant amount of elastic energy is stored in the myocyte and the interstitium as torsion*
- *The earliest mechanical manifestation of diastole is an abrupt untwisting that is largely completed before the mitral valve opens*
- *This untwisting helps to establish a base-to-apex intraventricular pressure gradient in early diastole that assists in the low pressure filling of the heart*
- *Modulation of this mechanism allows the heart to augment its function many-fold during exercise*



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