

# Ventricular-arterial coupling

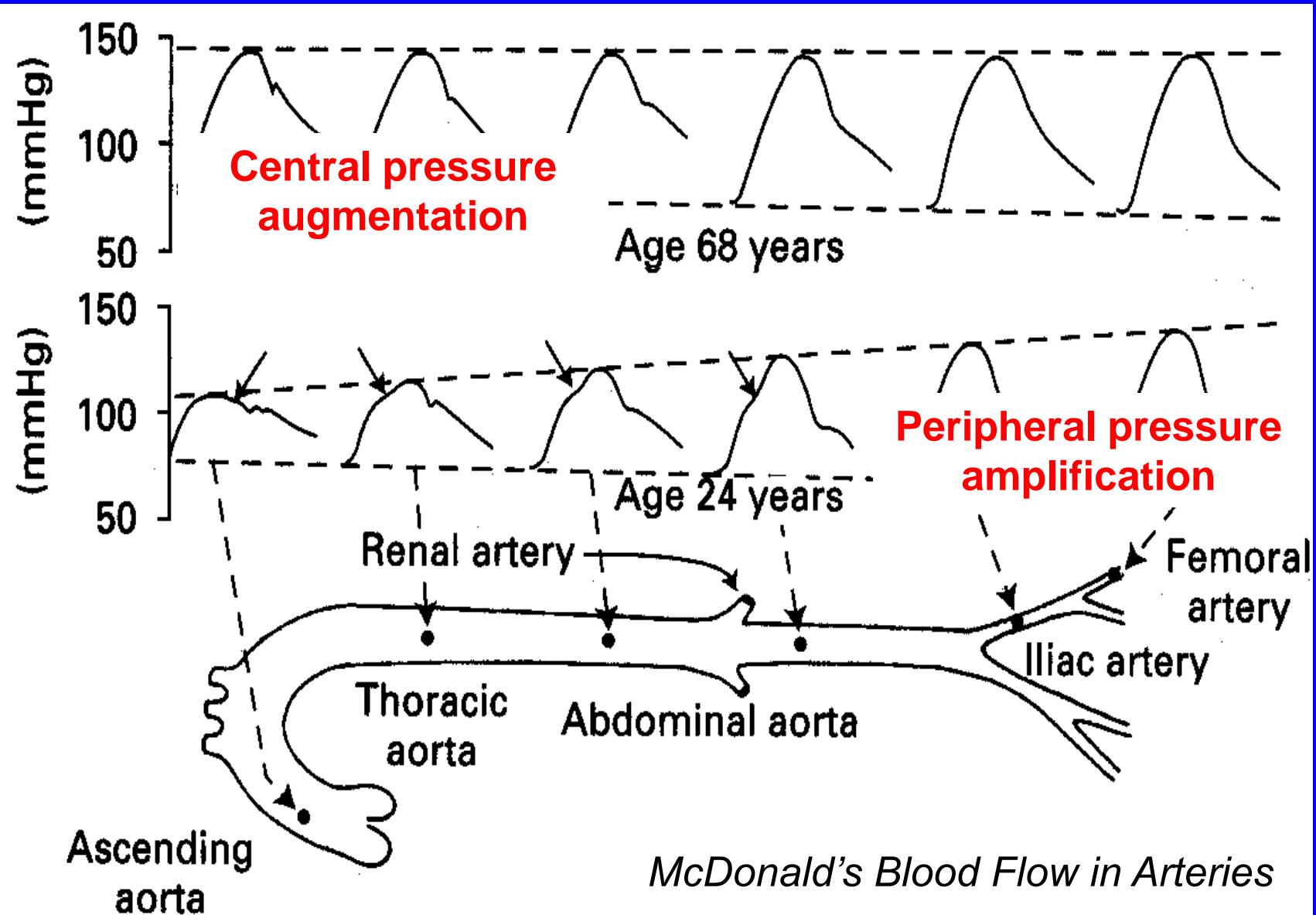
Prof. Dr. Alan Fraser  
Cardiff University

## Ventricular–arterial coupling

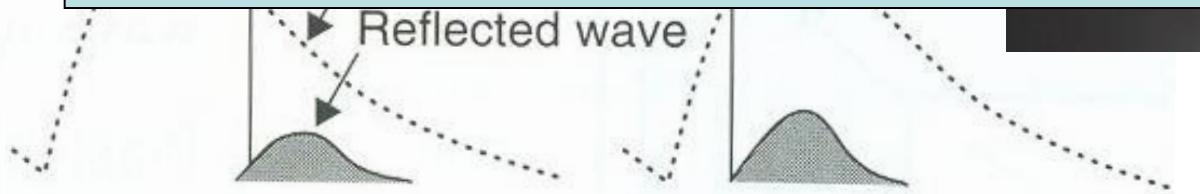
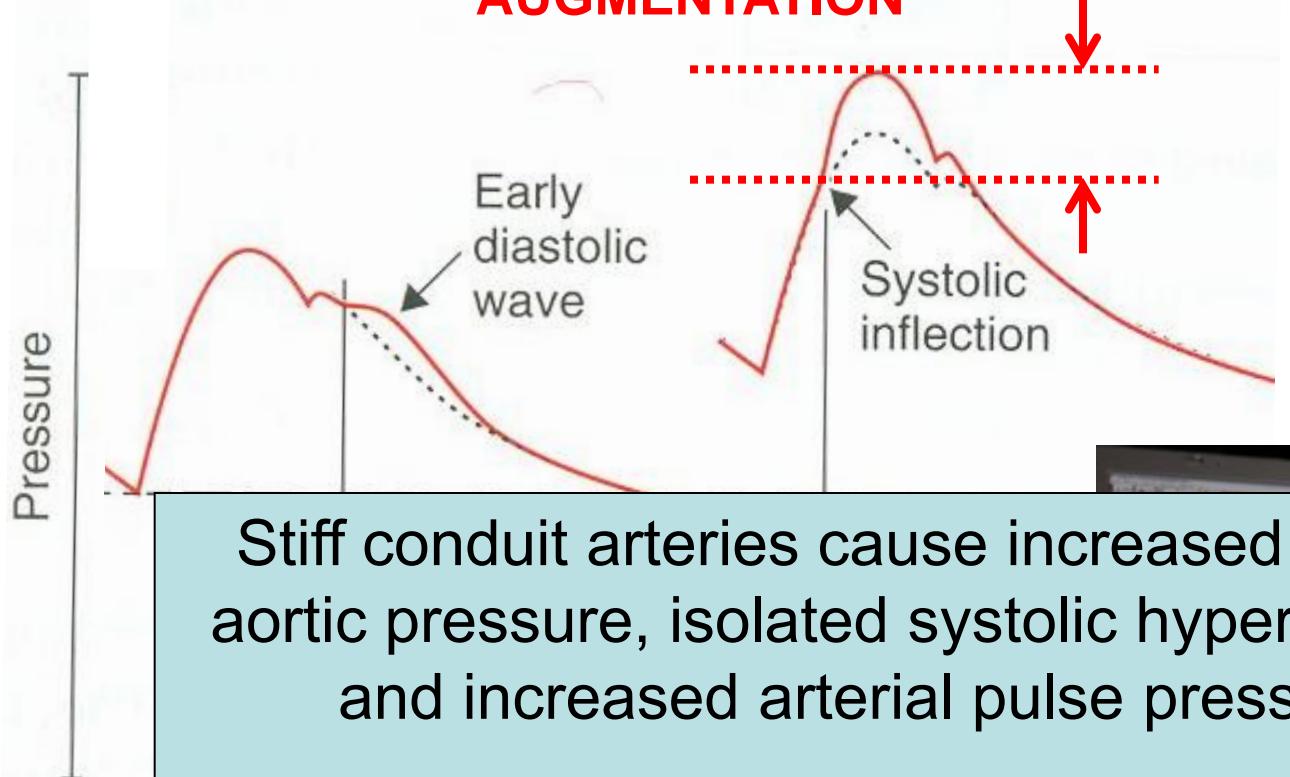
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- Increased loading triggers LV hypertrophy
- Ventricular response is asymmetrical
- Central arterial pressure
- Conduit arterial stiffness
- Ventricular and vascular elastance
- Arterial waves and energy

# Central arterial pressure changes with age



## AUGMENTATION



Applanation tonometry

# Imaging arterial stiffness

- **BETA INDEX**

- Pressure-independent Young's modulus of stiffness

- Adjusted for BP

- No units

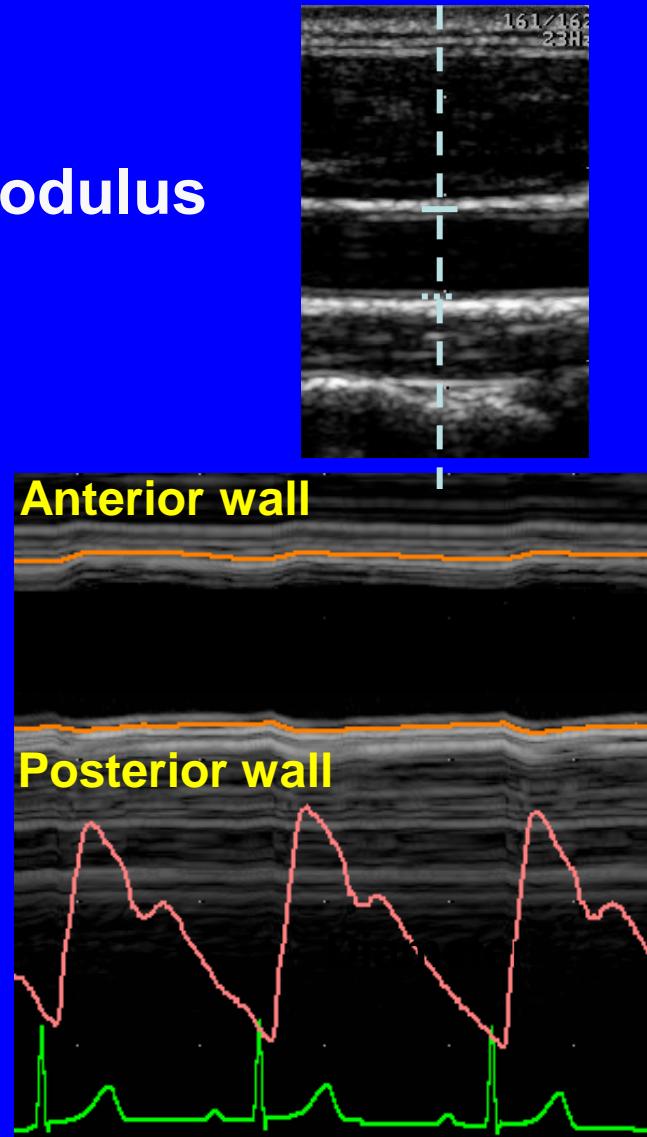
$$\beta = \frac{\log_n (P_s / P_d)}{(D_s - D_d / D_d)}$$

- **EPSILON**

- Pressure-strain elastic modulus

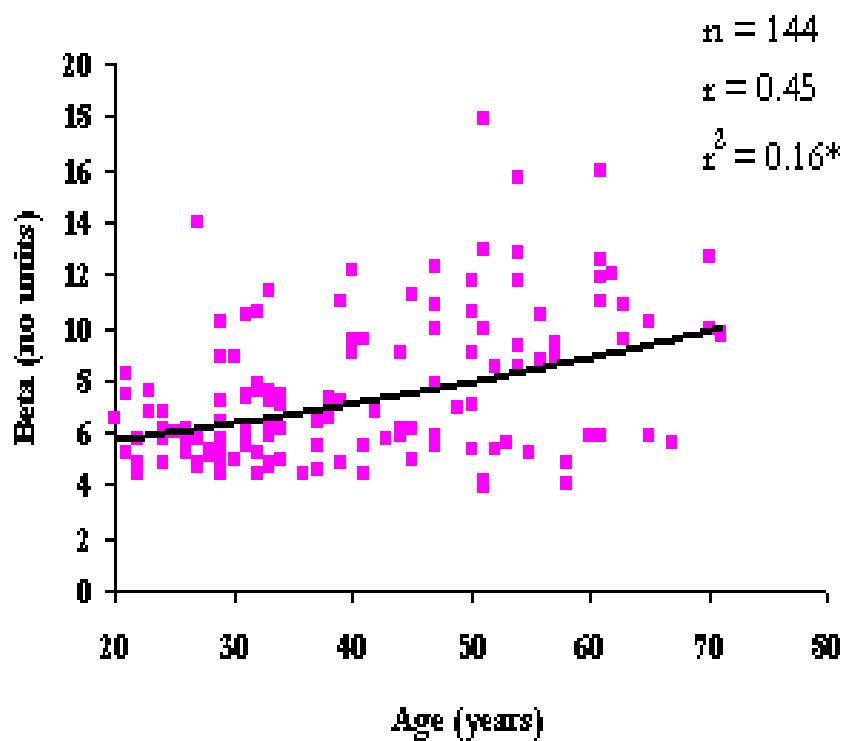
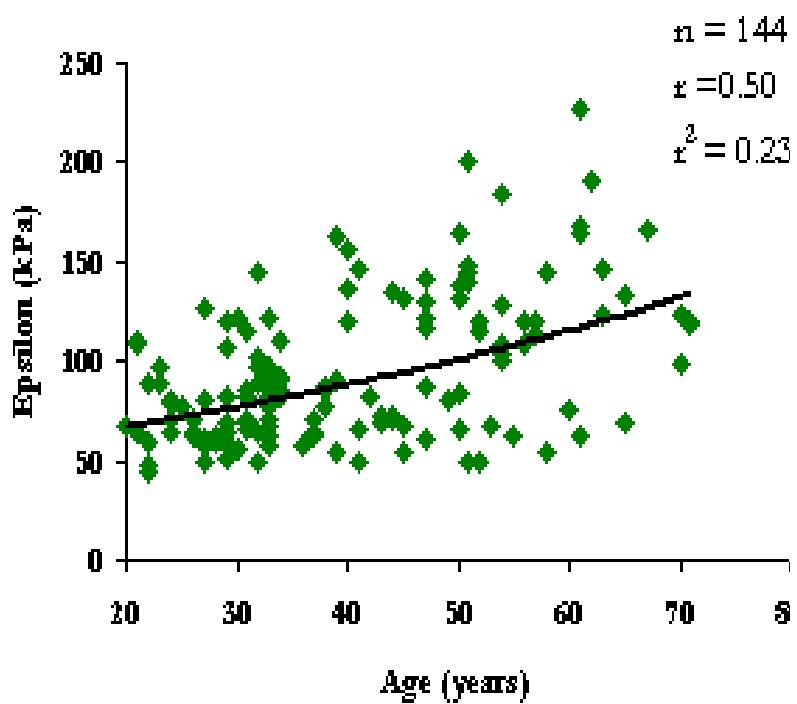
- Measured in kPa

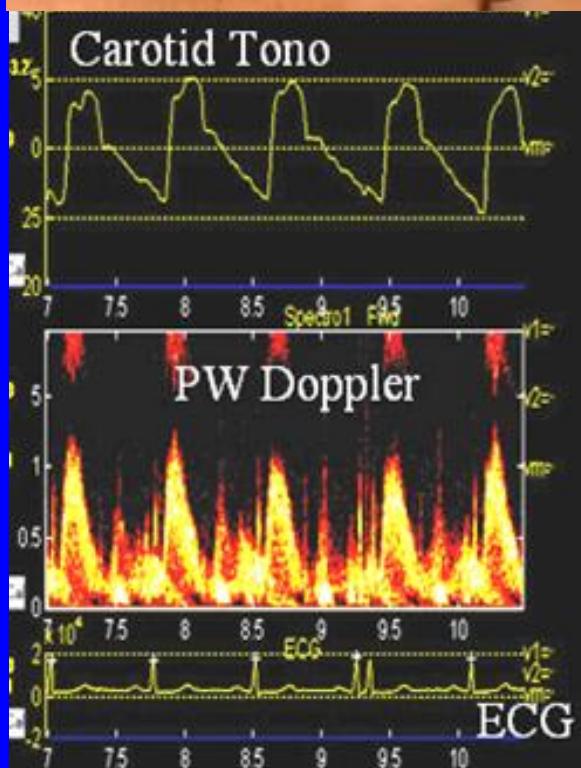
$$E_p = \frac{(P_s - P_d) D_d}{(D_s - D_d)}$$



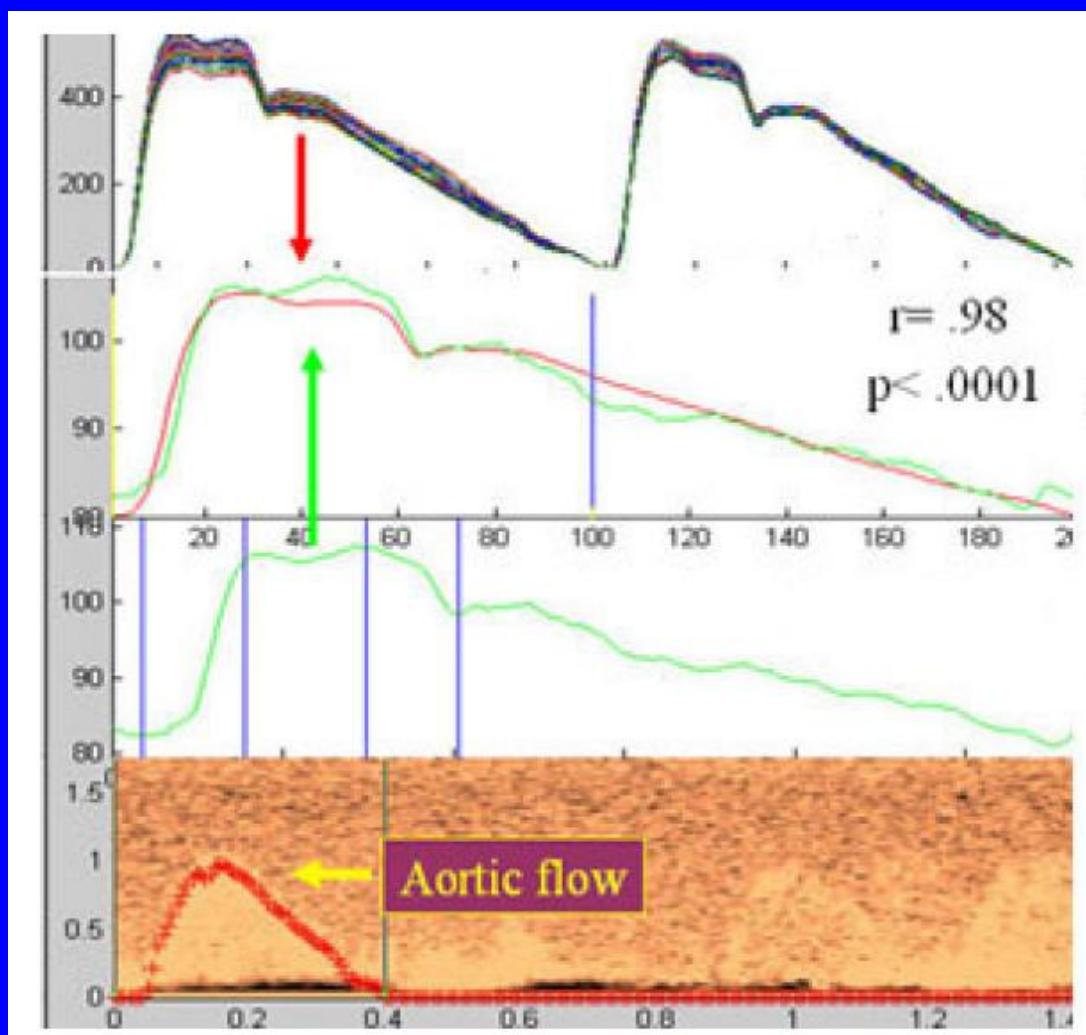
# Pooled observations in 144 subjects – Age

## Conduit arterial stiffness



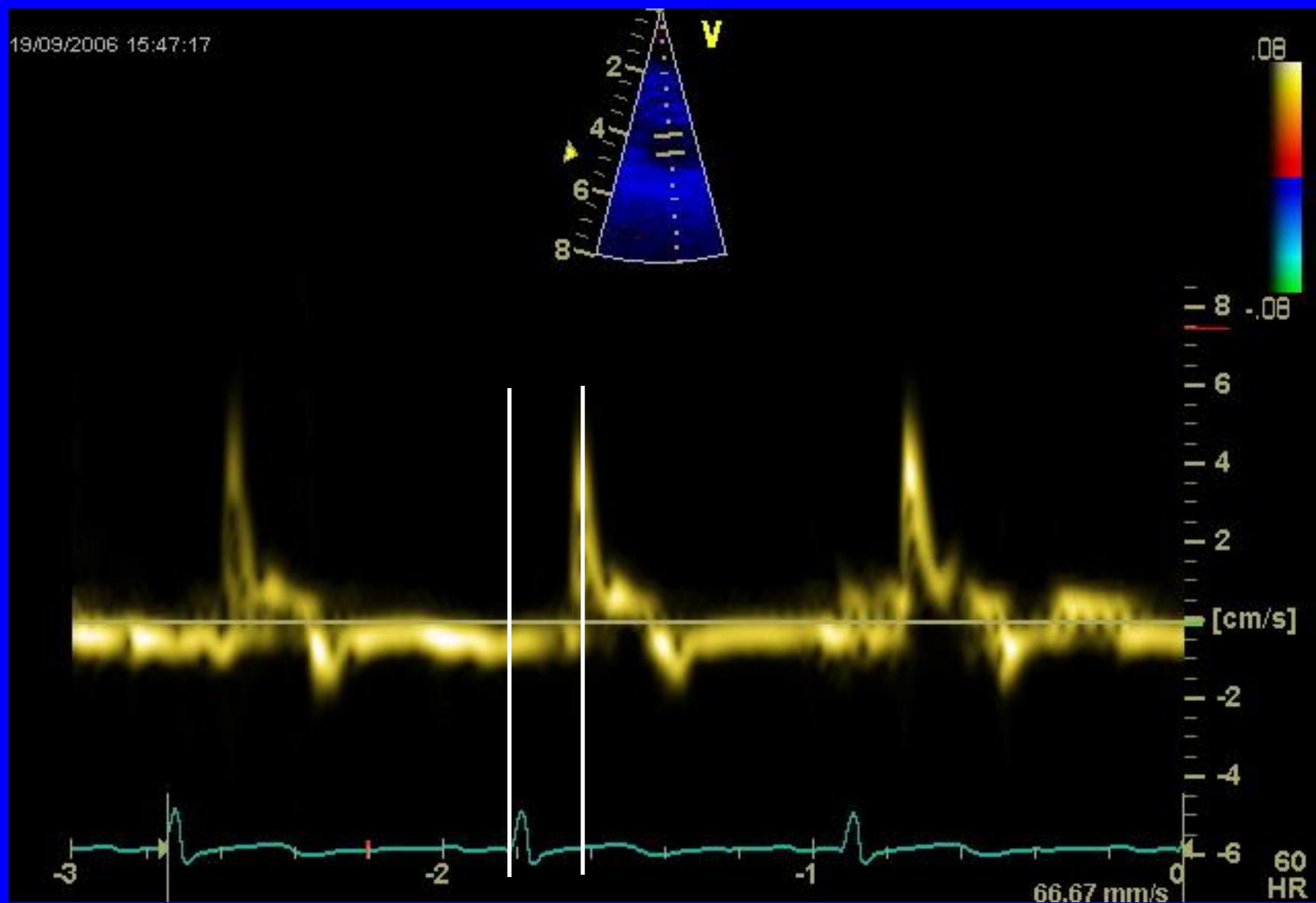


Central pressure waveforms can be derived from tissue Doppler



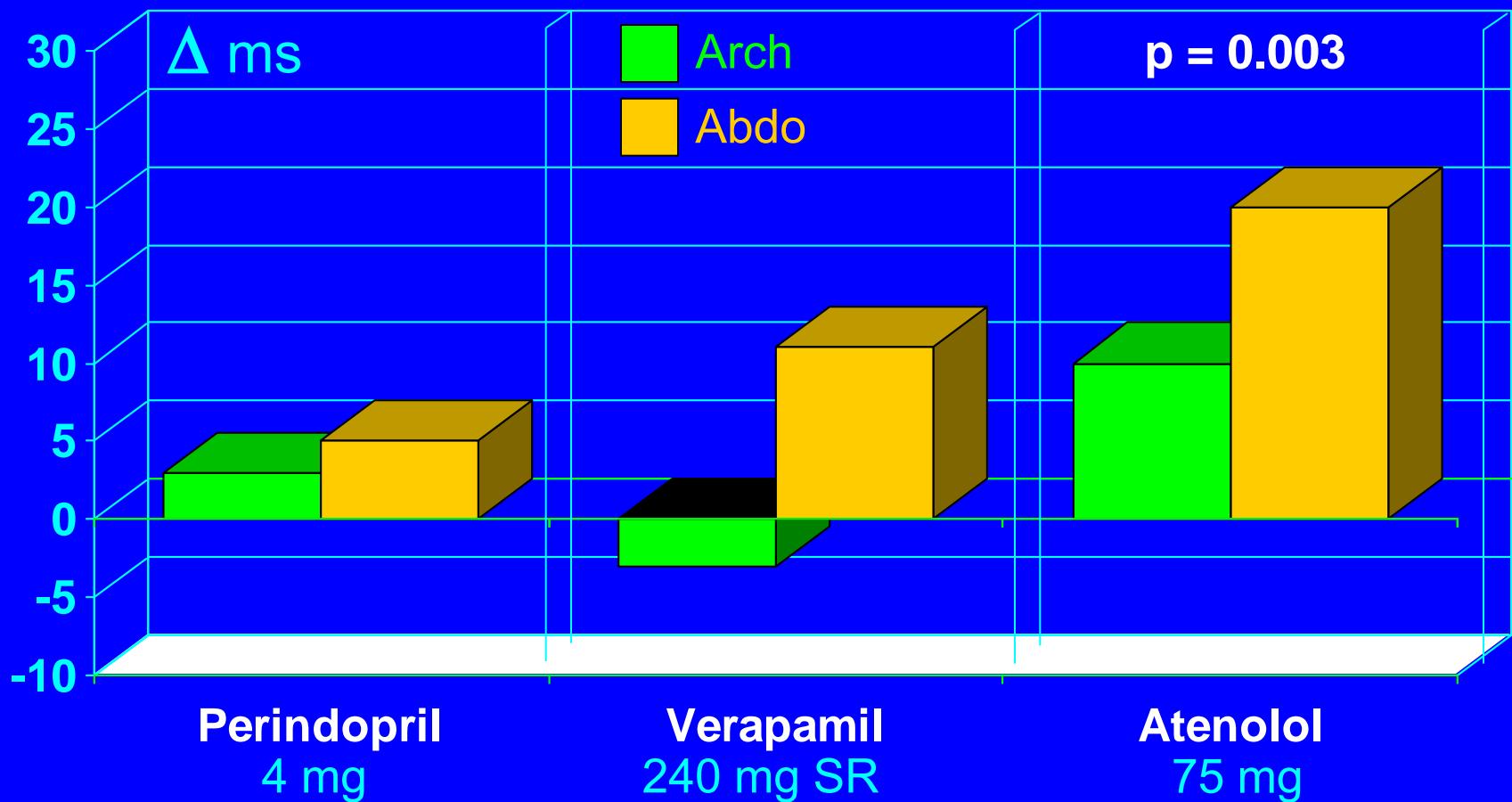
Haluska B et al, Cardiovasc Ultrasound 2007;5:6

# Tissue velocity of aortic wall motion



# Aortic wall motion in Marfan syndrome

## Prospective, double-blind, randomised cross-over

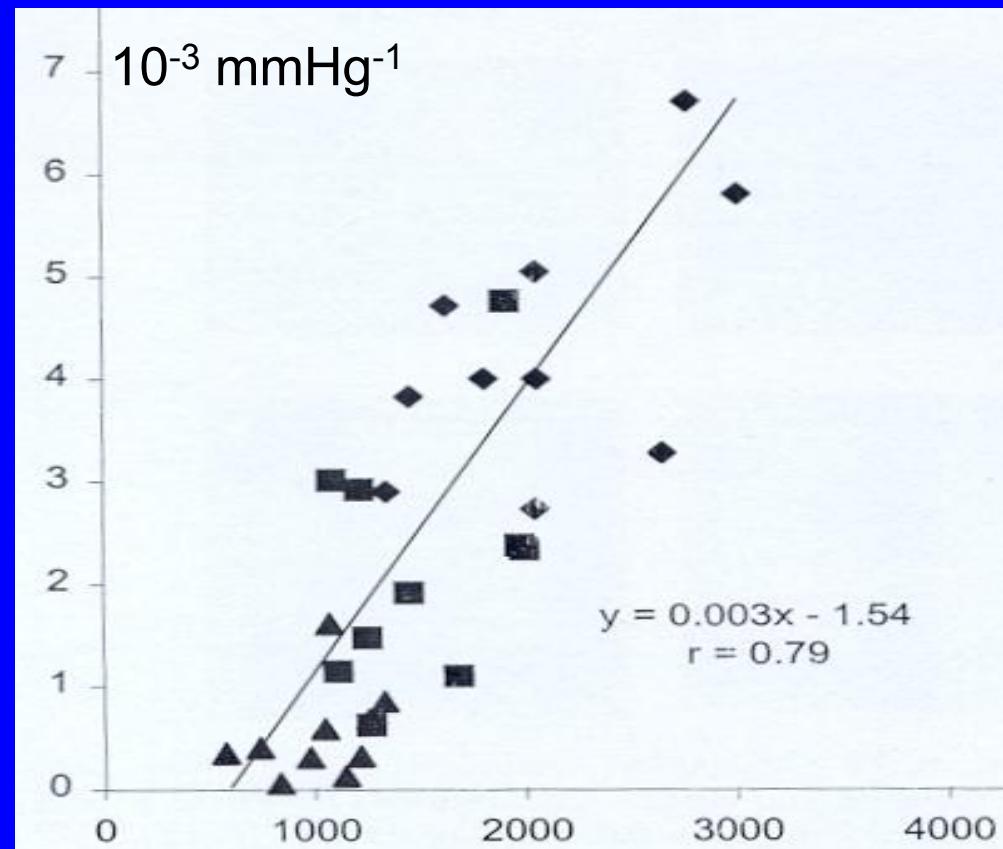


Time to peak systolic velocity  
of anterior aortic wall motion

Williams A et al

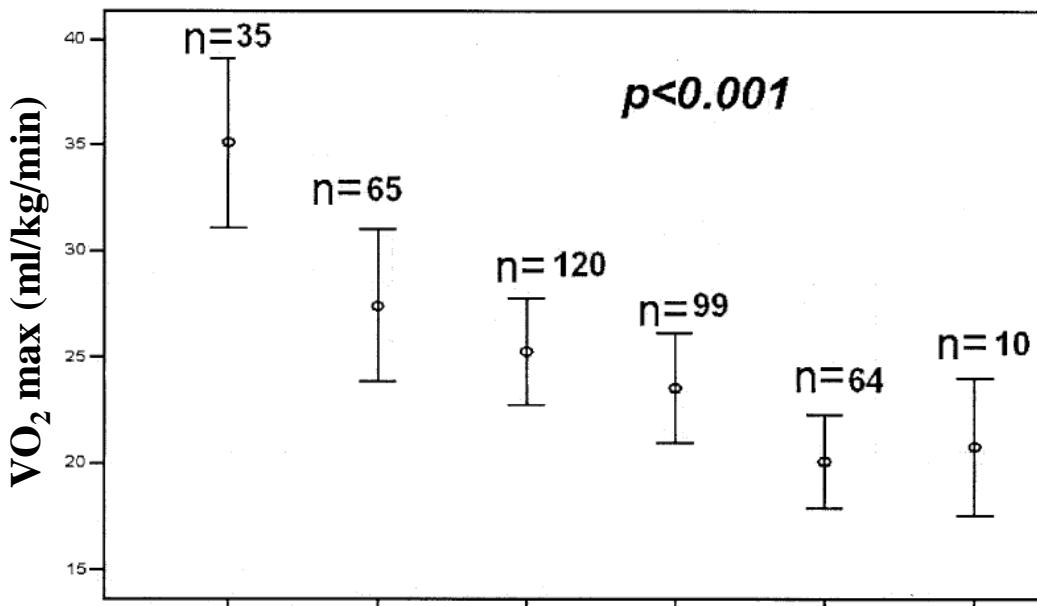
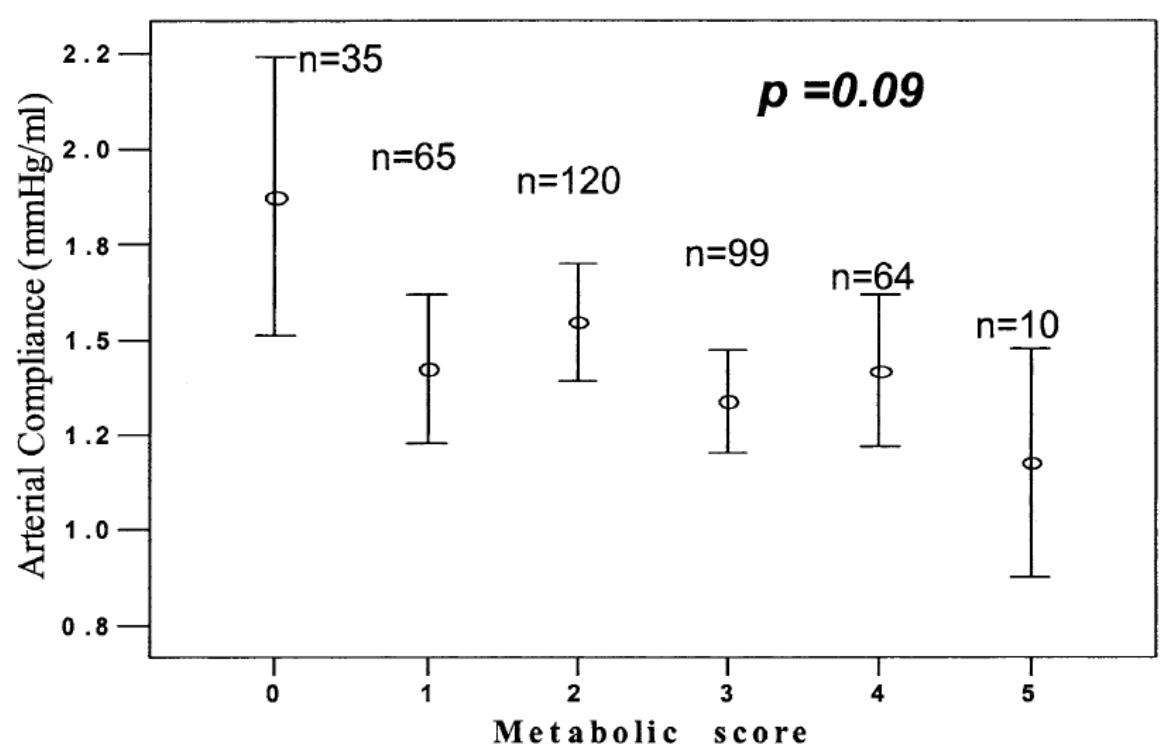
# Arterial stiffness is a determinant of $\text{VO}_2 \text{ max}$

Distensibility



$\text{VO}_2 \text{ max (ml/min)}$

Hundley, JACC 2001; 38: 796



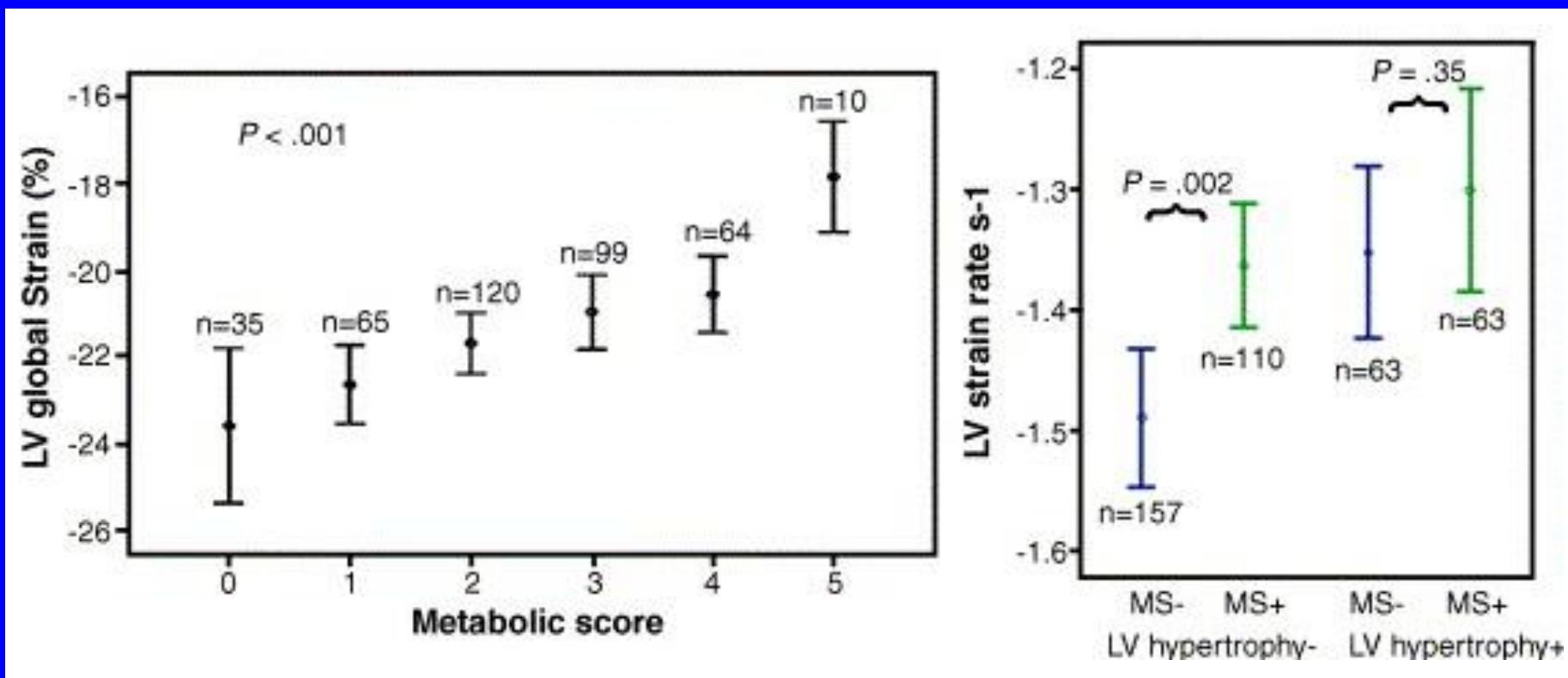
Arterial compliance & peak exercise capacity are related

& both decline with risk factors & severity of the metabolic syndrome

Wong C et al,  
AJC 2005;96:1686

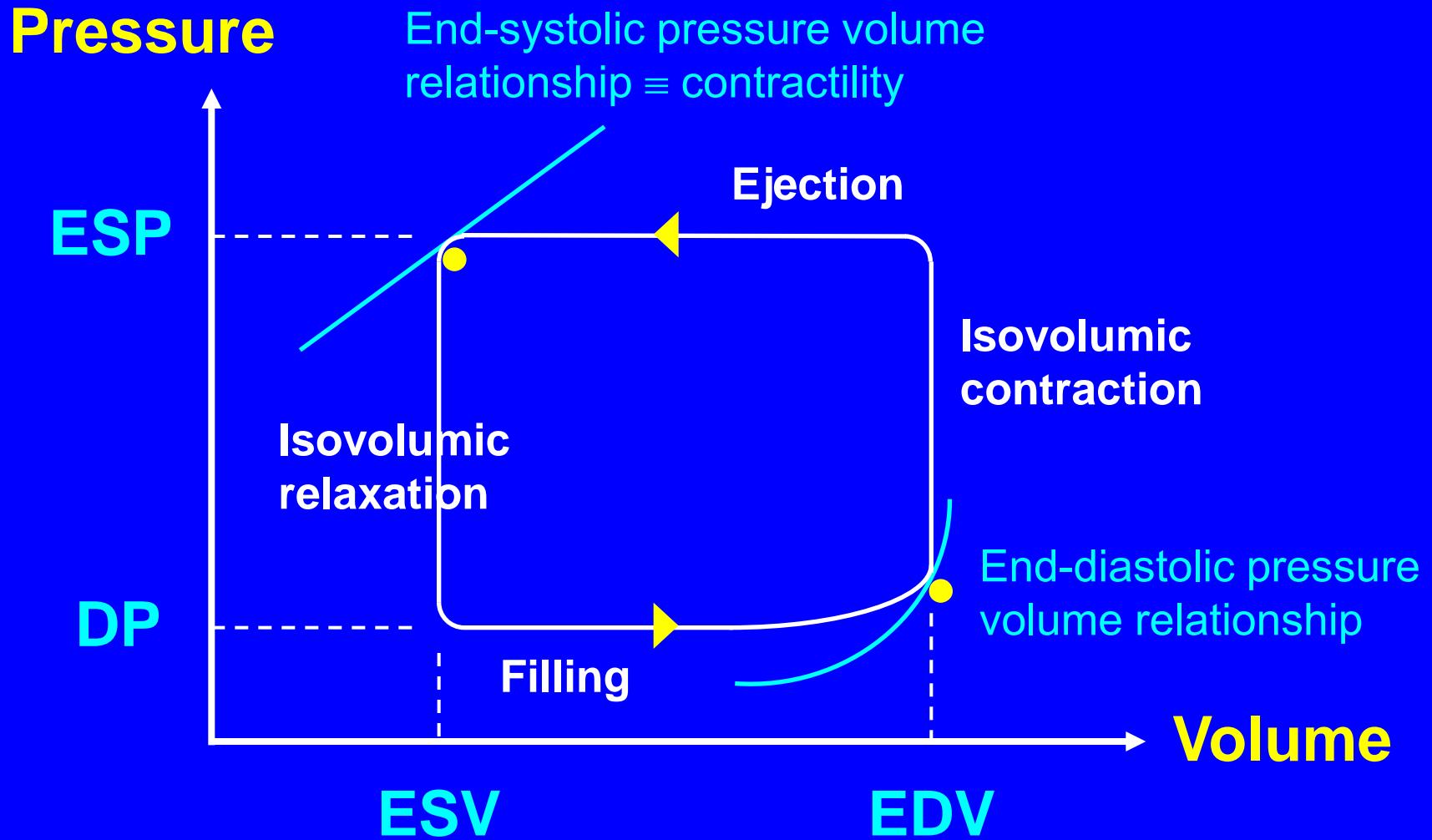
# Myocardial dysfunction in metabolic syndrome correlates with number of diagnostic features

393 subjects with negative stress echocardiography



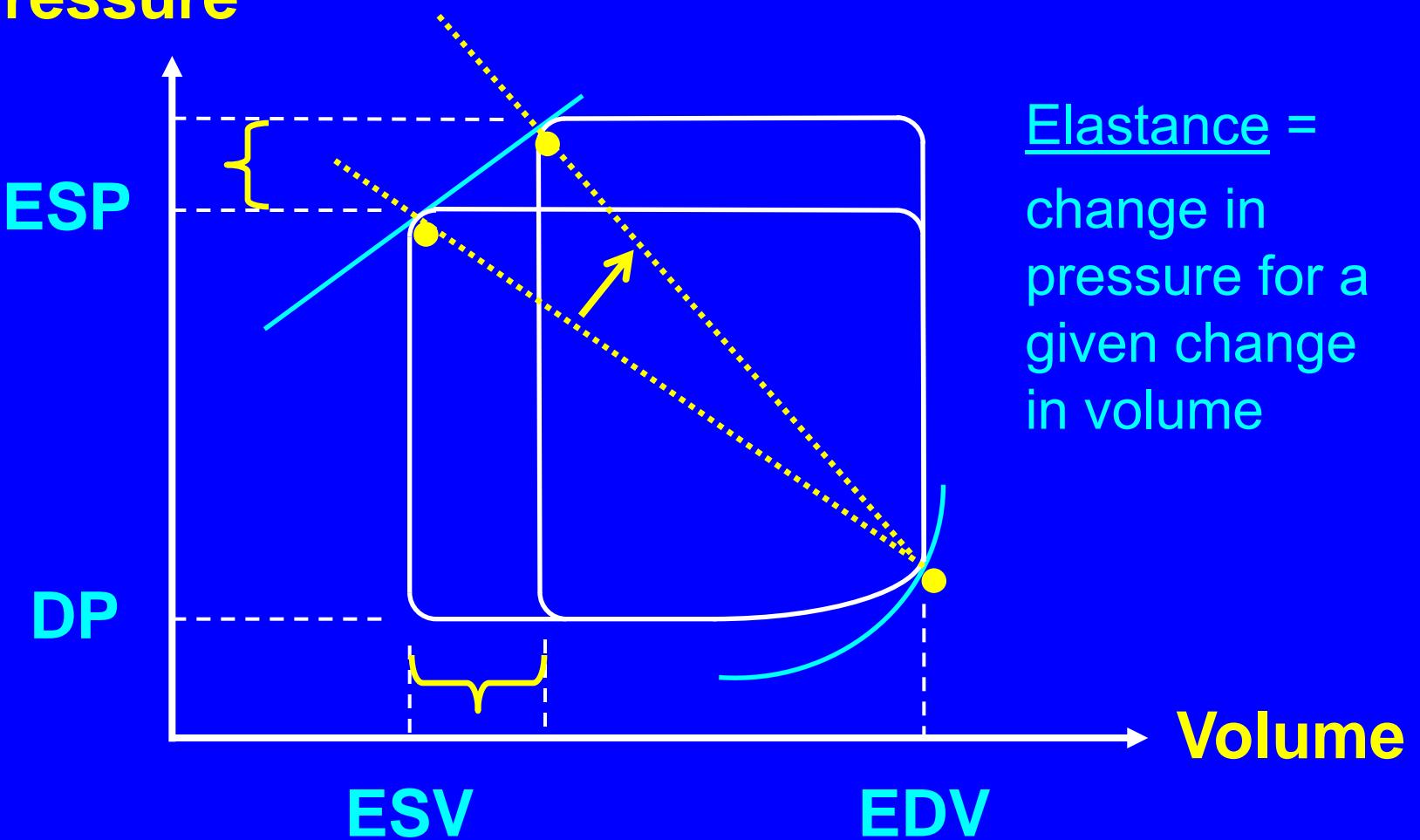
Wong C et al, Am J Cardiol 2005; 96: 1686-91

# Left ventricular pressure-volume loop



# The traditional concept of ventricular-arterial coupling

Pressure



# Elastance

Change in pressure for a unit change in volume  
(mmHg/ml)

- Arterial elastance

$$E_A = ESP / SV \text{ (SV stroke volume)}$$

Higher elastance = greater sensitivity to volume change, more variable pressure

- Ventricular elastance

$$E_{LV} = ESP / ESV \text{ (ESV LV end-systolic volume)}$$

Net arterial load exerted on the ventricle

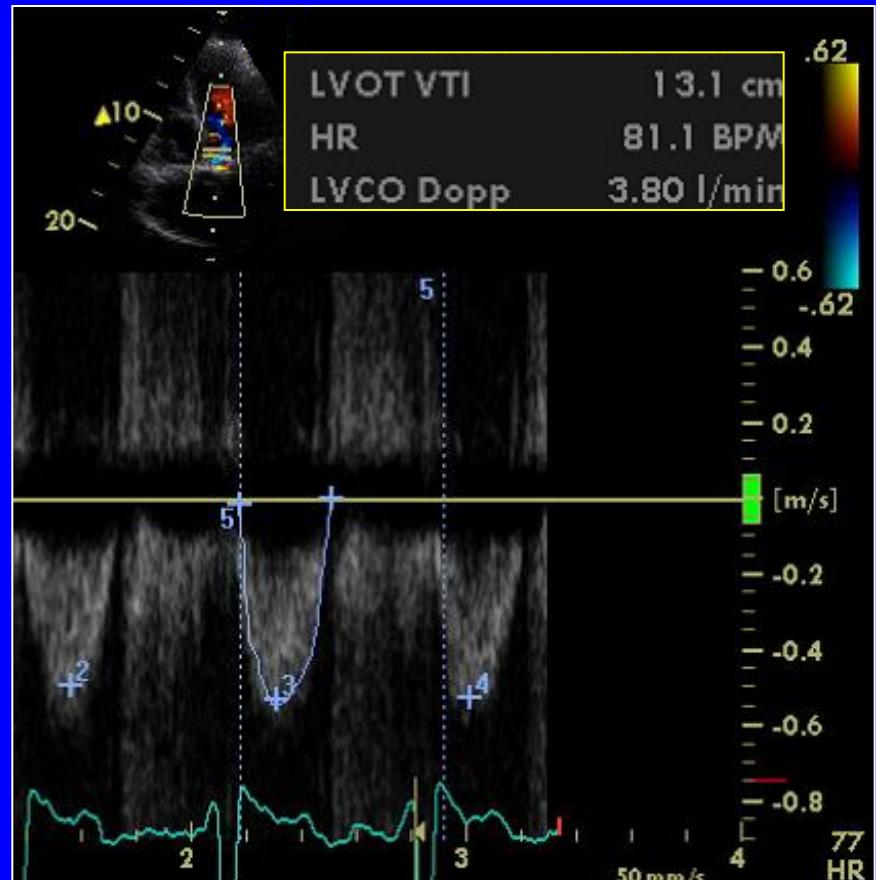
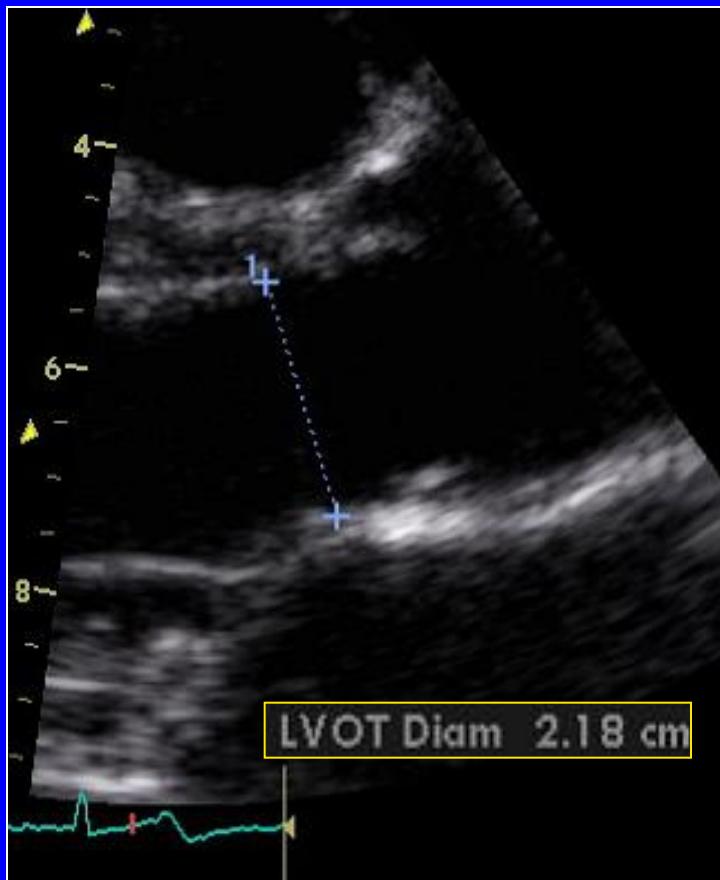
# Coupling ratio

$$VAC = E_A / E_{LV} \text{ with volumes indexed for body surface area}$$

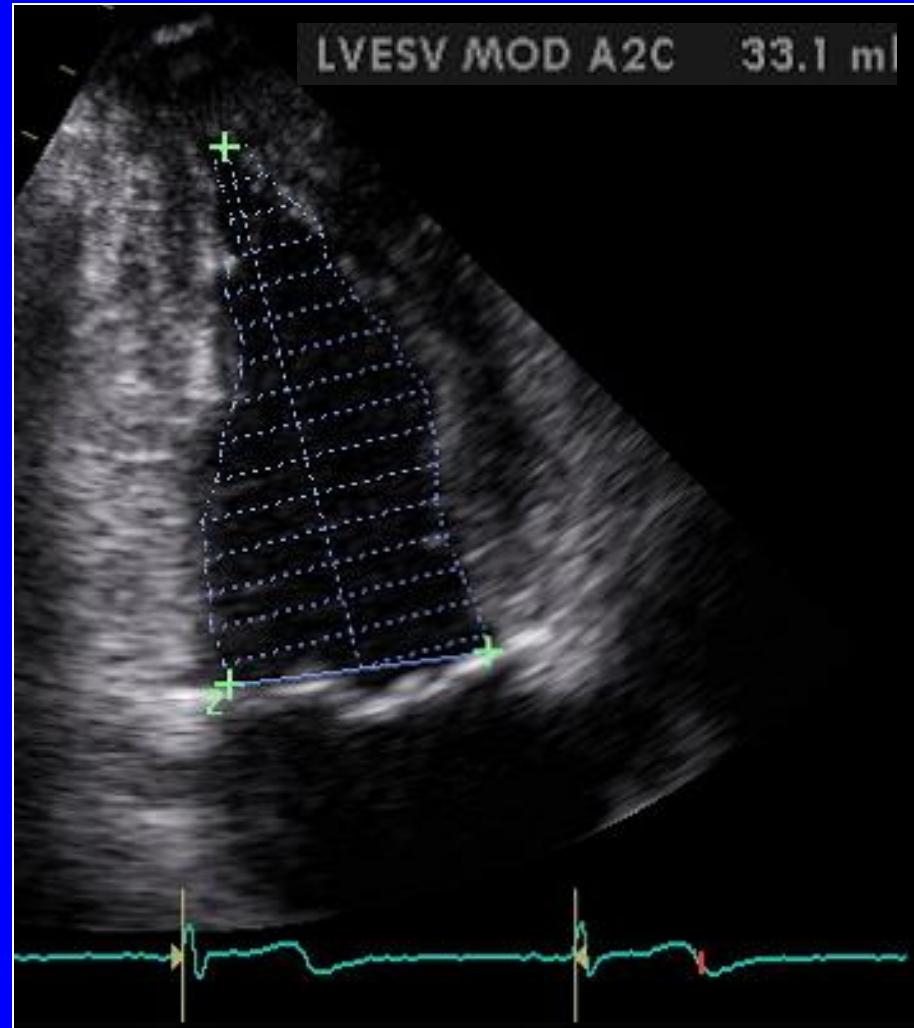
- Greatest efficiency when elastances are matched
- Optimal transfer of blood from LV to aorta
- BP, LV pressure, and CO are maintained in a physiological range
- Normal ratio  $\sim 1.0 \pm 0.36$  ( $VAC = (1/EF) - 1$ )
- Normal  $E_A 2.2 \pm 0.8 \text{ mmHg/ml}$
- Normal  $E_{LV} 2.3 \pm 1.0 \text{ mmHg/ml}$

*Chantler, 2008*

# M aged 30, triathlete



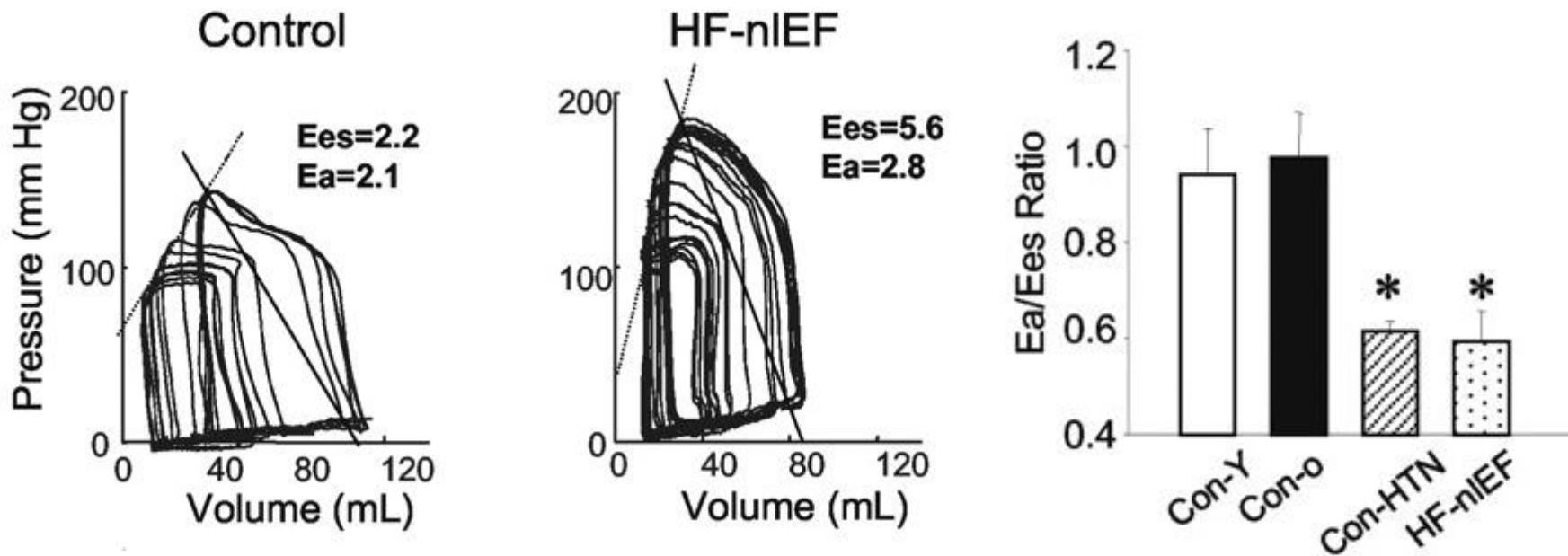
$$SV = 49 \text{ ml}$$



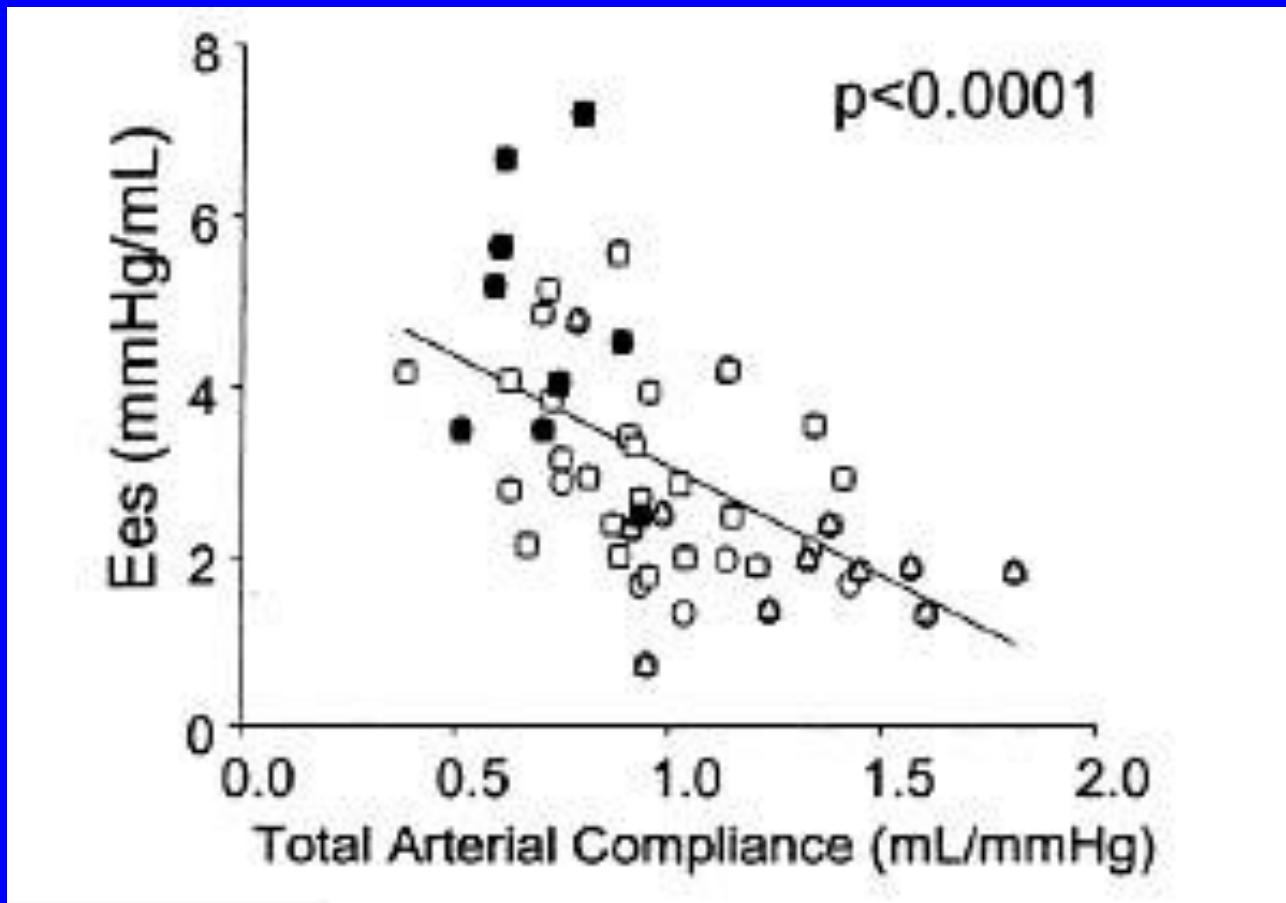
# Combined Ventricular Systolic and Arterial Stiffening in Patients With Heart Failure and Preserved Ejection Fraction

## Implications for Systolic and Diastolic Reserve Limitations

Miho Kawaguchi, MD; Ilan Hay, MD; Barry Fetics, MSE; David A. Kass, MD



# Myocardial contractility in the left ventricle is inversely related to arterial compliance



Kawaguchi M et al, Circulation 2003; 107: 714-20

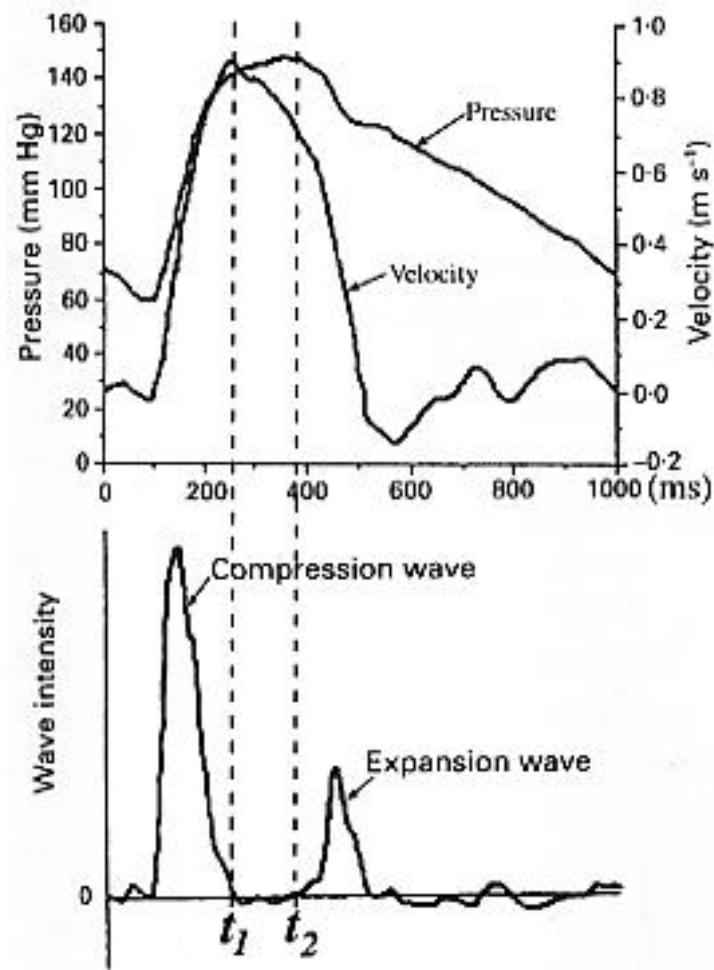
# Forward and Backward Running Waves in the Arteries: Analysis Using the Method of Characteristics

K. H. Parker

C. J. H. Jones

Physiological Flow Studies Unit,  
Imperial College of Science, Technology and  
Medicine,  
London, SW7 2AZ England

*Journal of Biomechanical  
Engineering* 1990; 112: 323



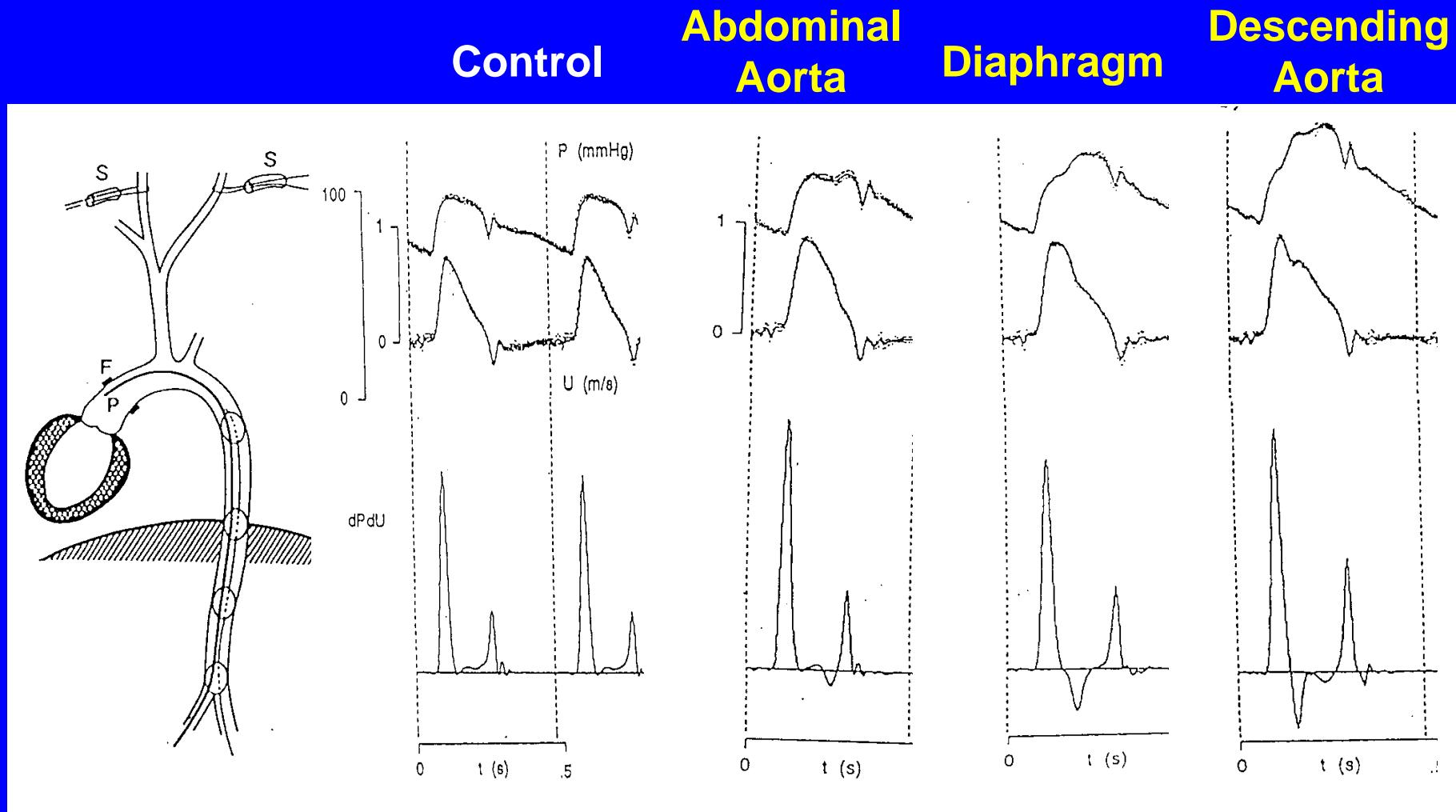
$$\text{Wave intensity} = \frac{dP}{dt} \cdot \frac{dU}{dt}$$

# There are 4 types of arterial waves

	Velocity	Pressure
Forward compression wave	↑	↑
Forward expansion wave	↓	↓
Backward compression wave	↓	↑
Backward expansion wave	↑	↓

*The integral of wave intensity is energy*

# Wave intensity and reflection



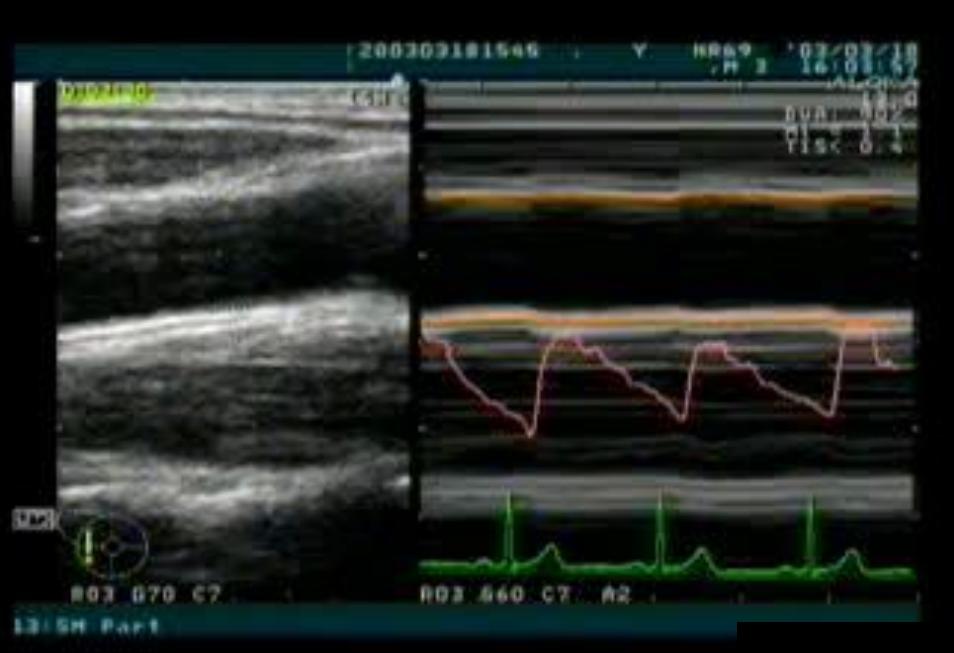
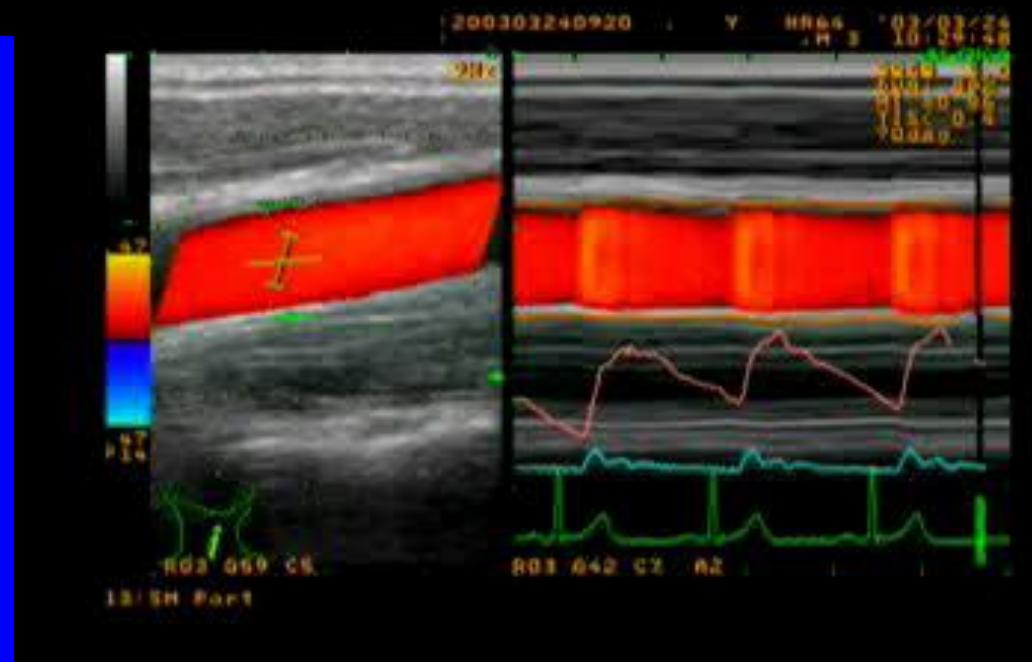
Ramsey, Jones & Sugawara

Wall tracking of common carotid artery

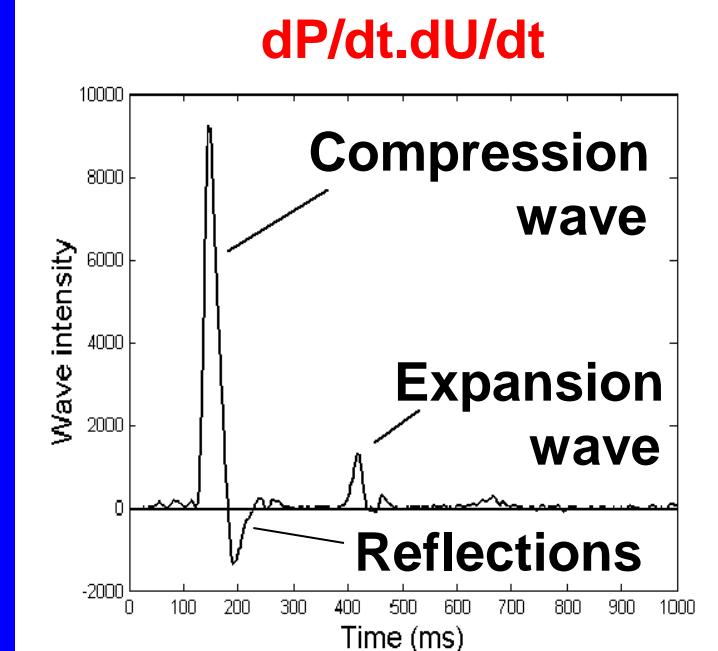
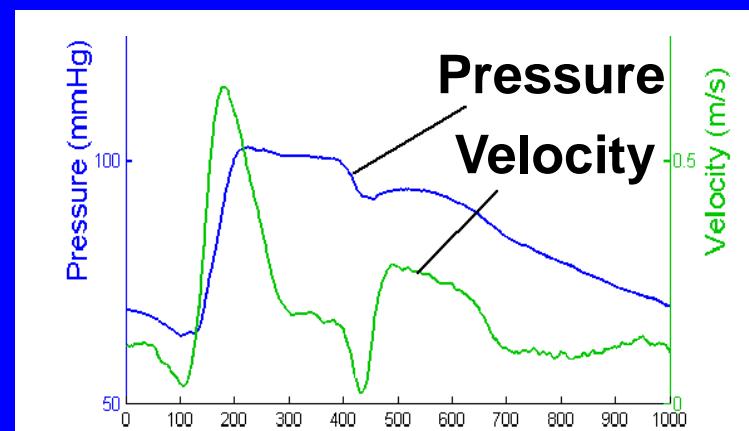
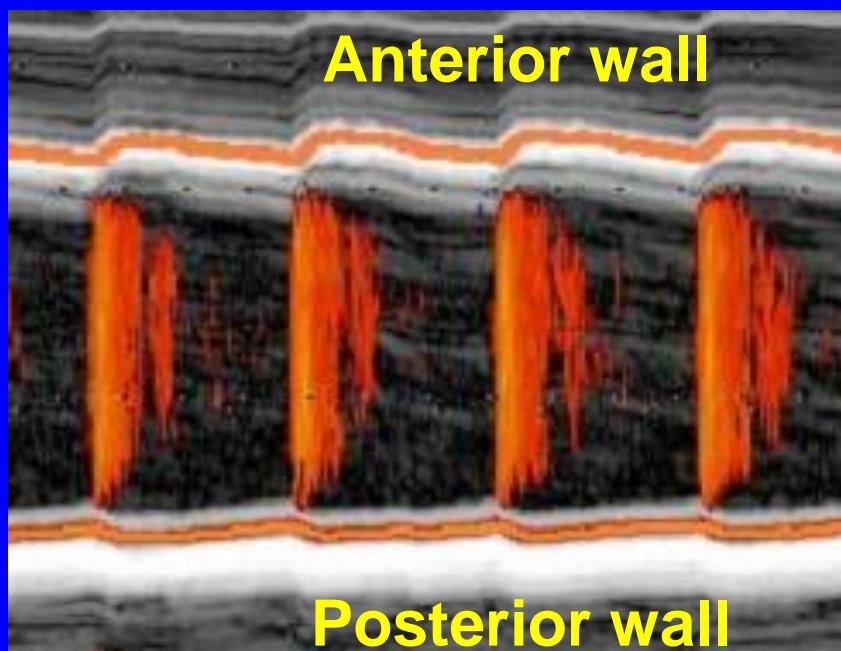
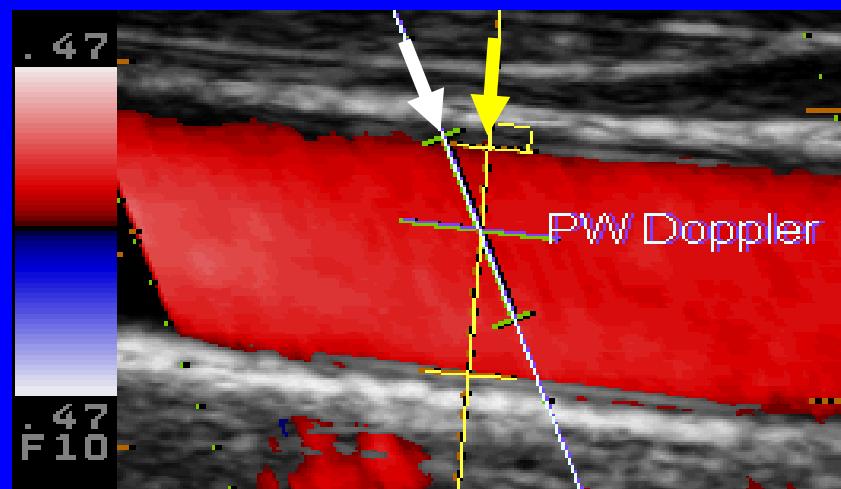
Diameter / distension waveform, calibrated as estimate of pressure

Simultaneous measurement of blood velocity at same site

→ Non-invasive wave intensity



# Aloka SSD 5500 7.5 MHz linear array transducer



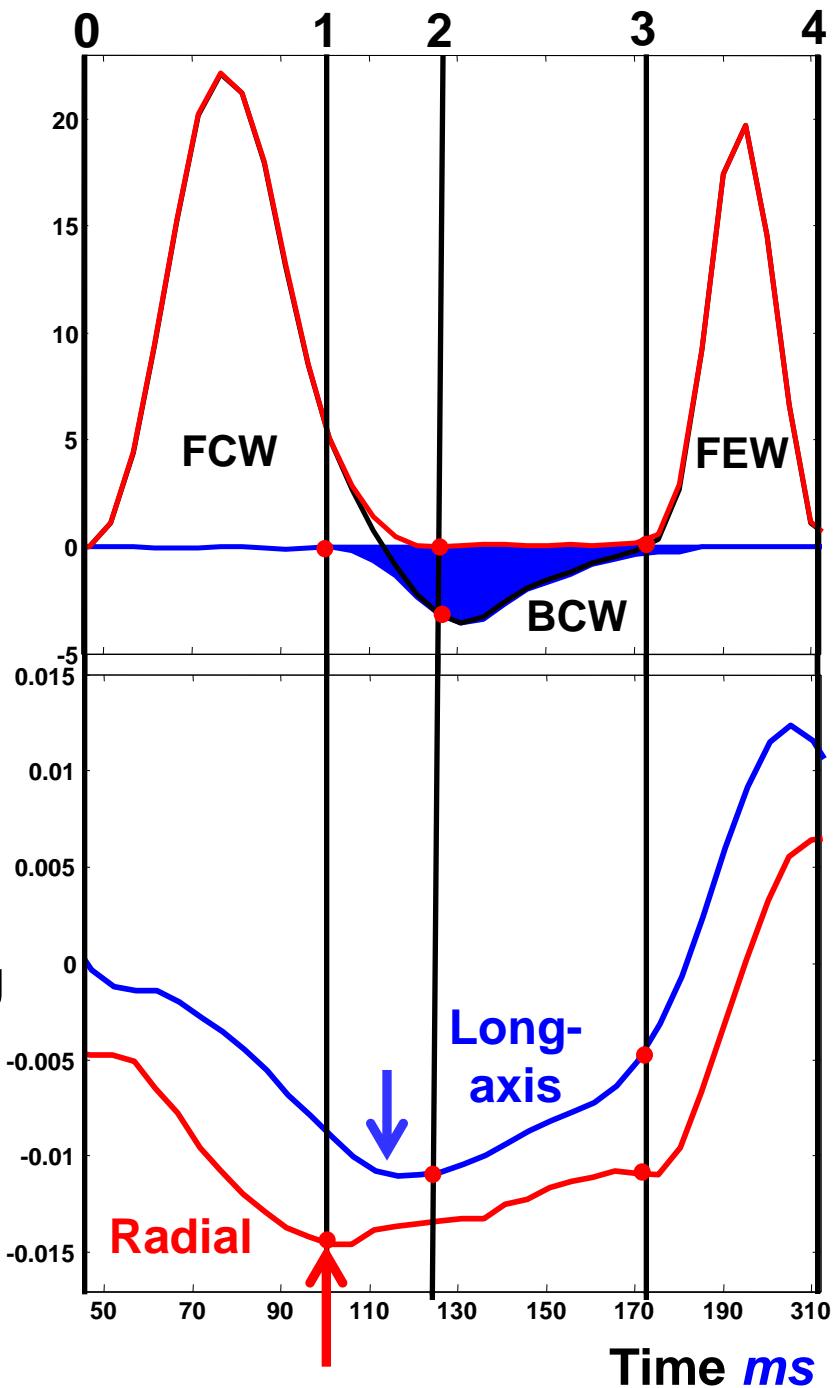
## Ventricular-arterial coupling

Conduit arterial stiffness impairs long-axis function

	Beta index	Pressure-strain elastic modulus	Integral of mid-systolic wave reflections
<i>Longitudinal S</i>	-0.45**	-0.37**	-0.37**
<i>Longitudinal E</i>	-0.53**	-0.47**	-0.50**
<i>Radial S</i>	-0.07	-0.02	-0.06
<i>Radial E</i>	-0.28*	-0.25*	-0.13
<i>Ejection fraction</i>	-0.21	-0.16	-0.15
<i>E/Ea</i>	0.48**	0.50**	0.34**

Wave intensity in the ascending aorta  
 $W/m^2$

Velocity of shortening  
 $m/s$

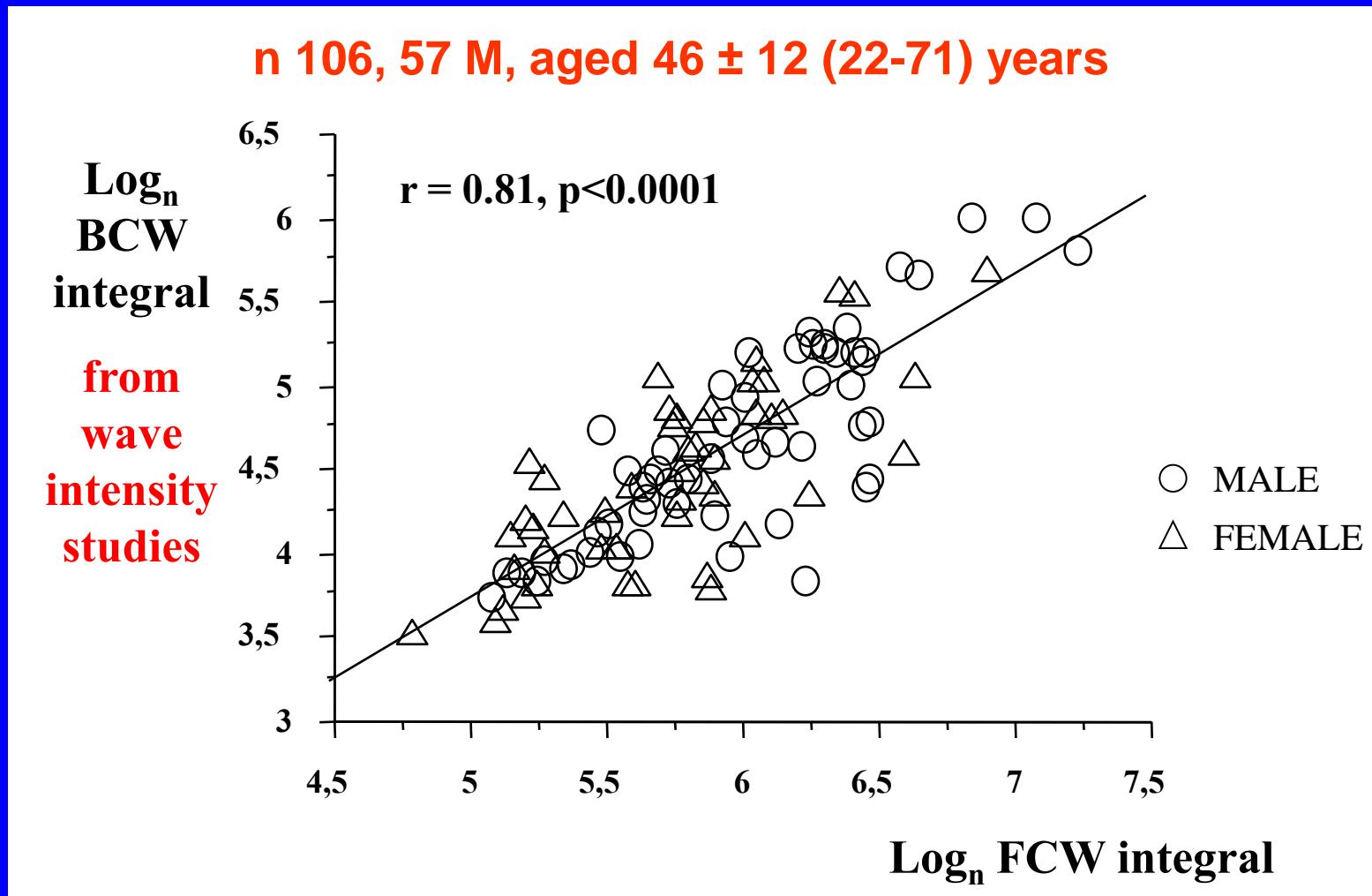


1. Peak velocity of radial shortening of the left ventricle coincides with arrival of reflected waves

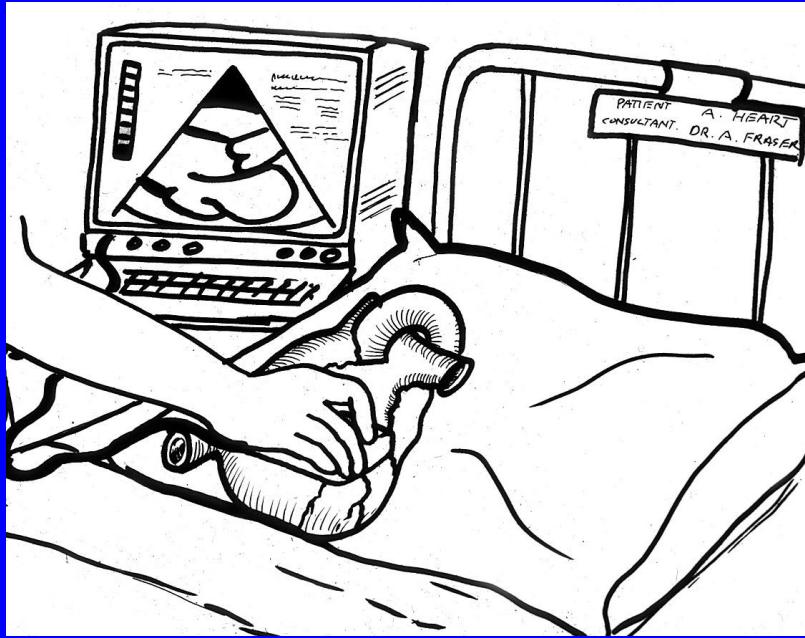
2. Generation of longitudinal shortening persists against reflections

Page C et al,  
Int J Cardiol 2009 e pub

# The major “determinant” of reflected wave energy is left ventricular systolic function



Rakebrandt F et al, Ultrasound Med Biol 2009; 35: 266-77



The heart does not operate independently ..  
.. it's part of an integrated cardiovascular symptom

## Why do we have to understand V-A coupling ?

- to understand mechanisms of LV dysfunction
- because conduit arterial stiffness, central arterial pressure, and wave reflections can be important new therapeutic targets