Ventricular-arterial coupling – clinical tools and diagnostic indications

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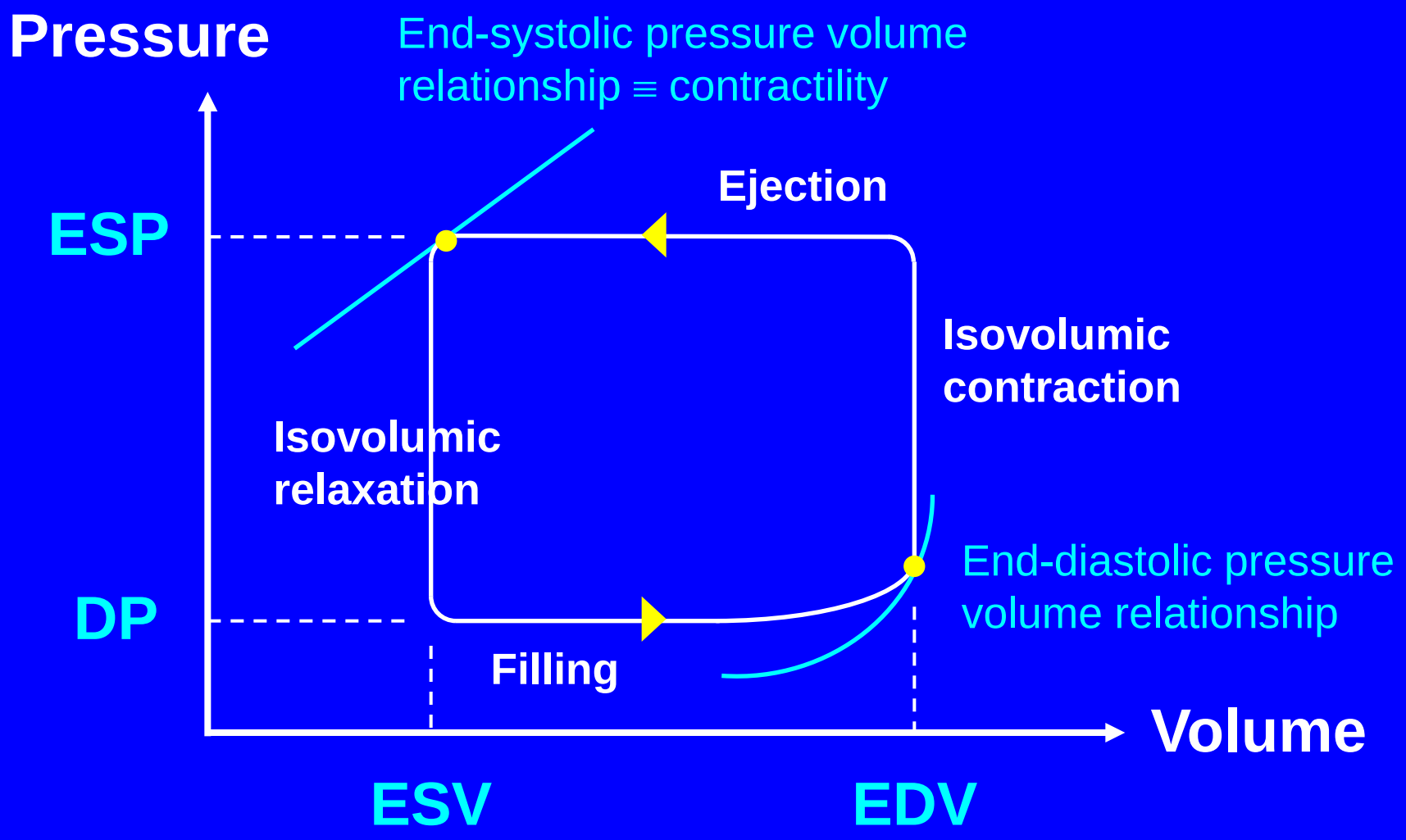
Support for research from Hitachi Aloka, & GE Ultrasound

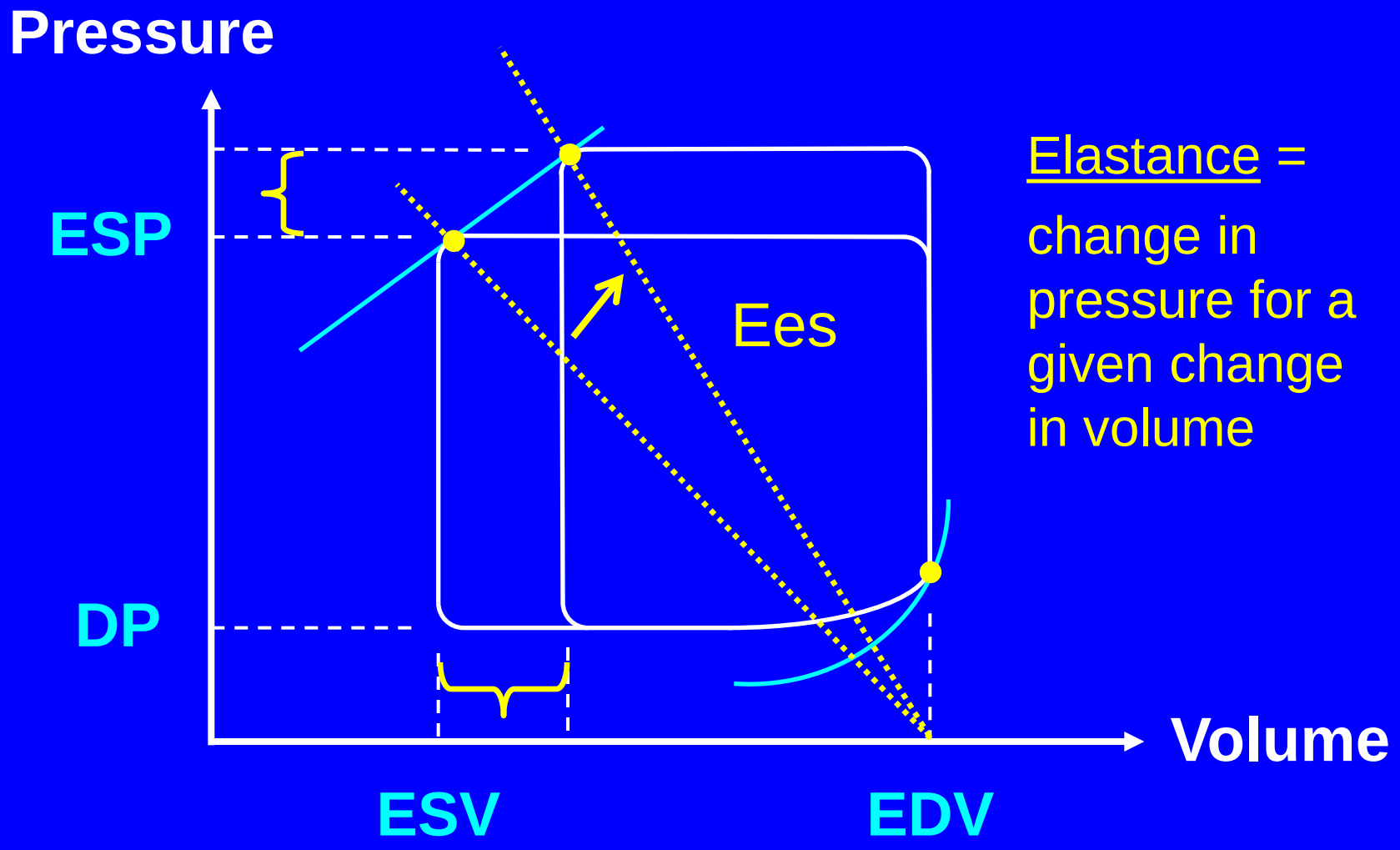
Ventricular–arterial coupling

- Ventricular and aortic elastances
- Local arterial stiffness
- Central aortic pressure
- Aortic pulse wave velocity
- Wave intensity, energy, and reflections
- Aortic wall motion and deformation
  - velocity & timing of systolic expansion
  - speckle tracking

Left ventricular pressure-volume loop

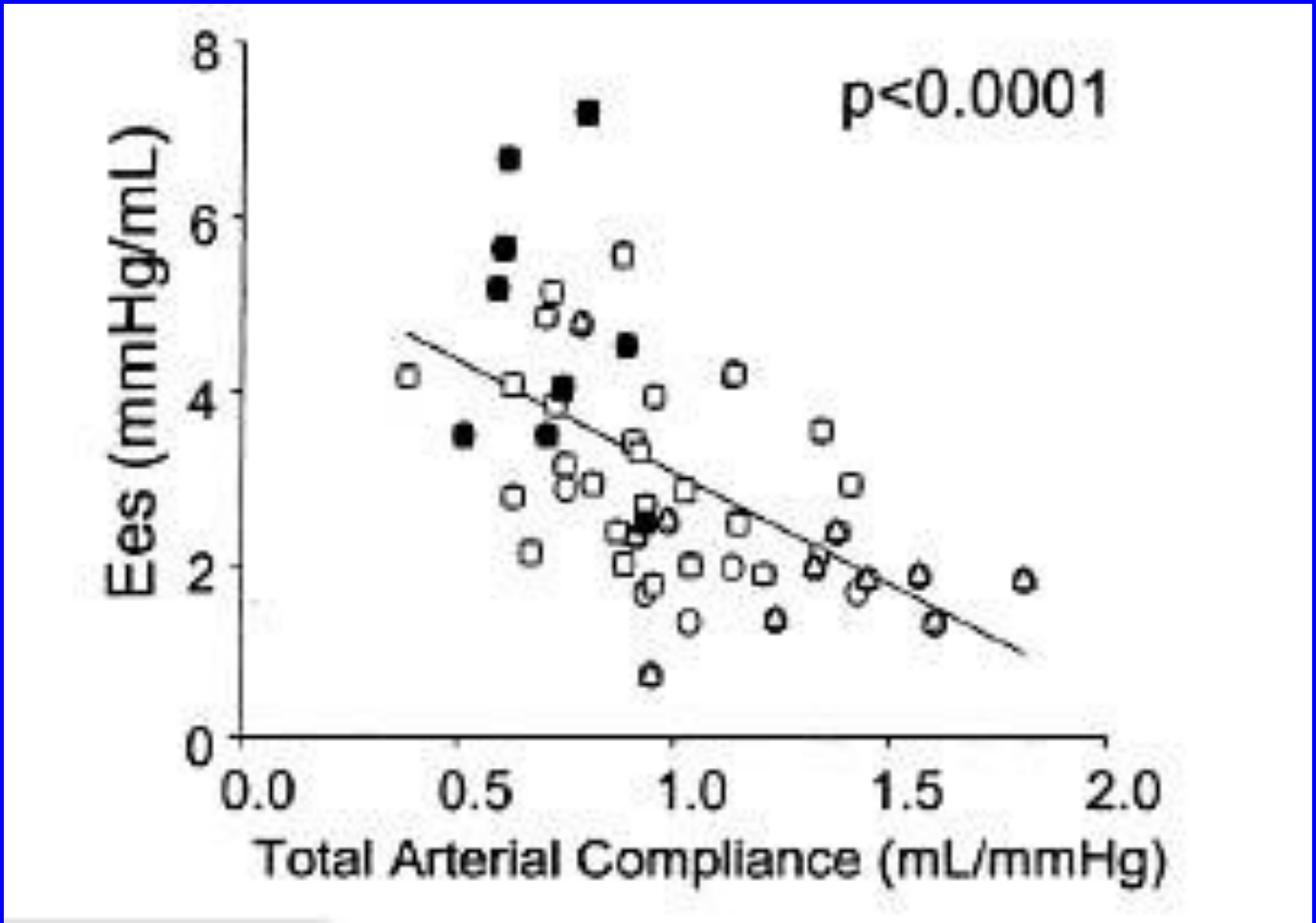


The “traditional” concept of ventricular-arterial coupling (Sunagawa)



Elastance = change in pressure for a given change in volume

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



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

Elastance

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

- Arterial elastance

  E_A = ESP / SV (SV stroke volume)

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

- Ventricular elastance (Ees)

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

  Net arterial load exerted on the ventricle

Ventricular-arterial coupling – ratio of elastances

VAC = E_A / E_{LV} 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 ~ 1.0 ± 0.36
- Normal E_A 2.2 ± 0.8 mmHg / ml
- Normal E_{LV} 2.3 ± 1.0 mmHg / ml

Chantler PD et al, J Appl Physiol 2008;105:1342-51

Ventricular-arterial coupling – example

$$VAC = E_A / E_{LV}$$

$$= ESP / SV \div ESP / ESV$$

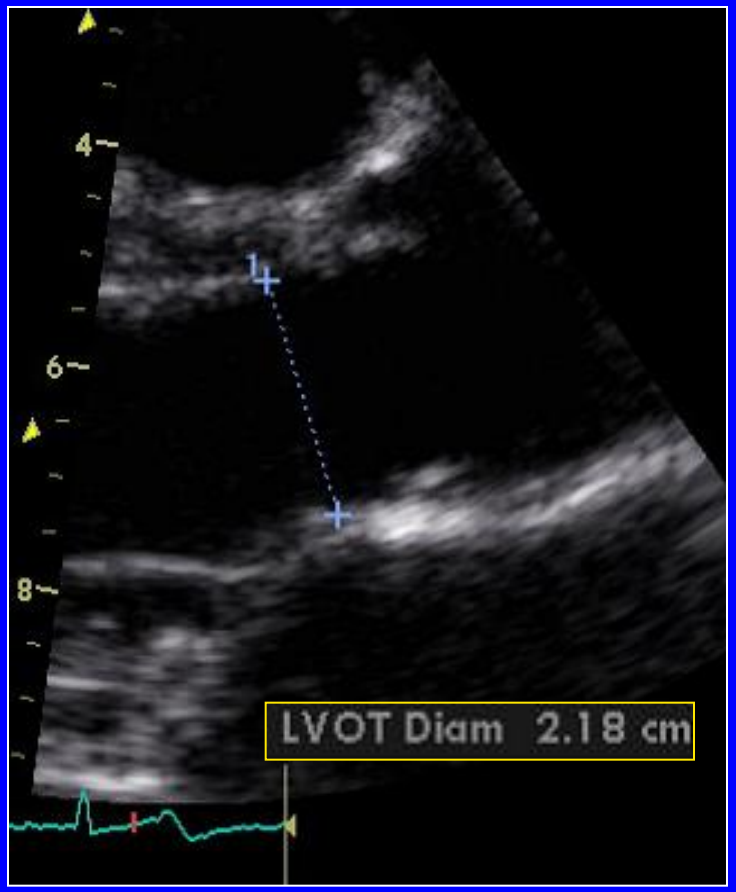
$$= ESP / SV \times 1 / (ESP / ESV)$$

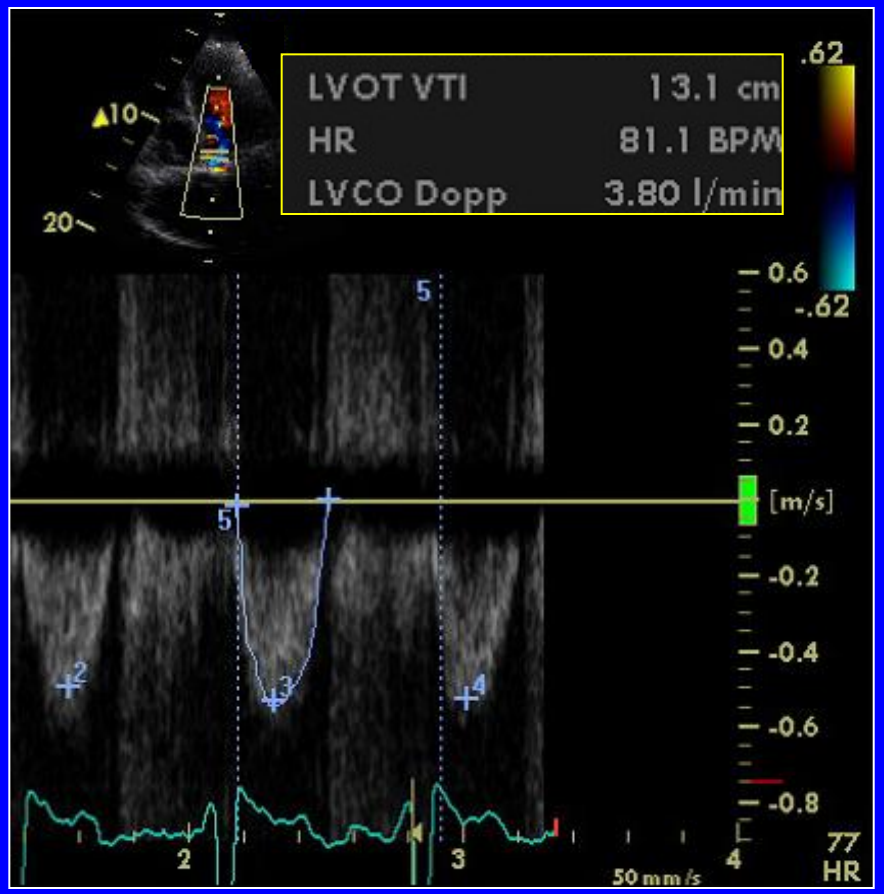
$$= ESP / SV \times ESV / ESP$$

$$\frac{\cancel{ESP}}{SV} \times \frac{ESV}{\cancel{ESP}}$$

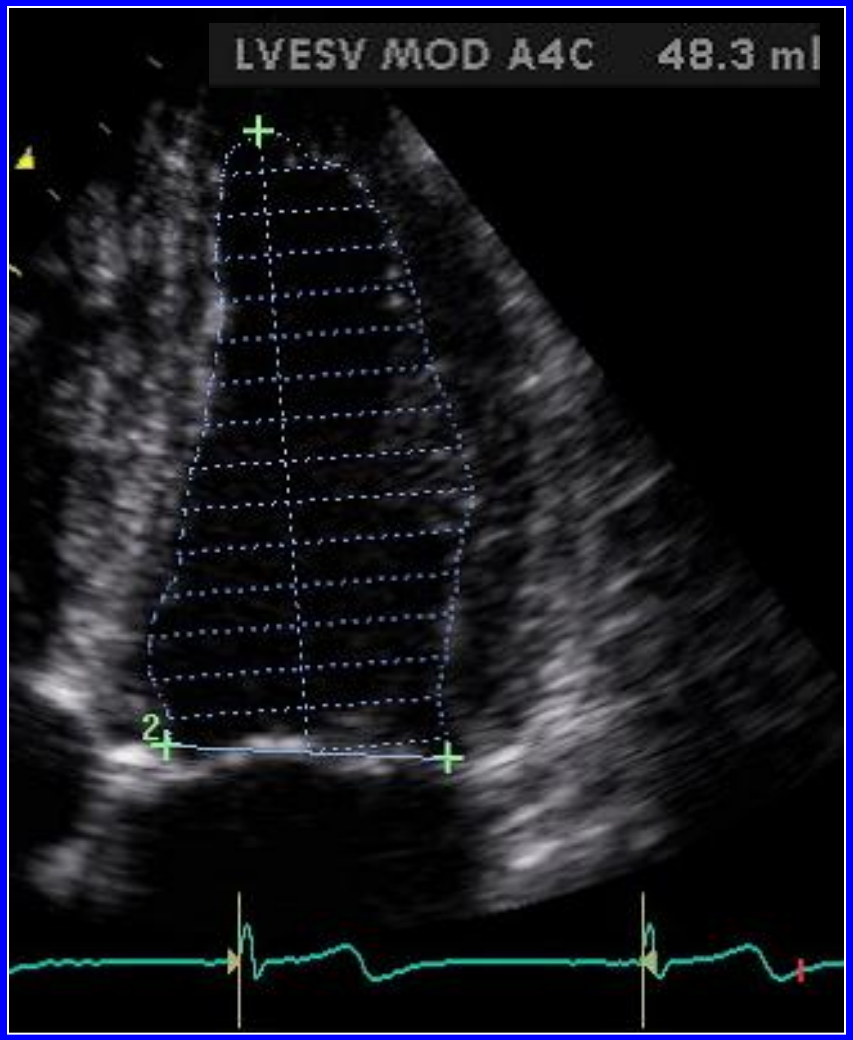
$$= ESV \div SV$$

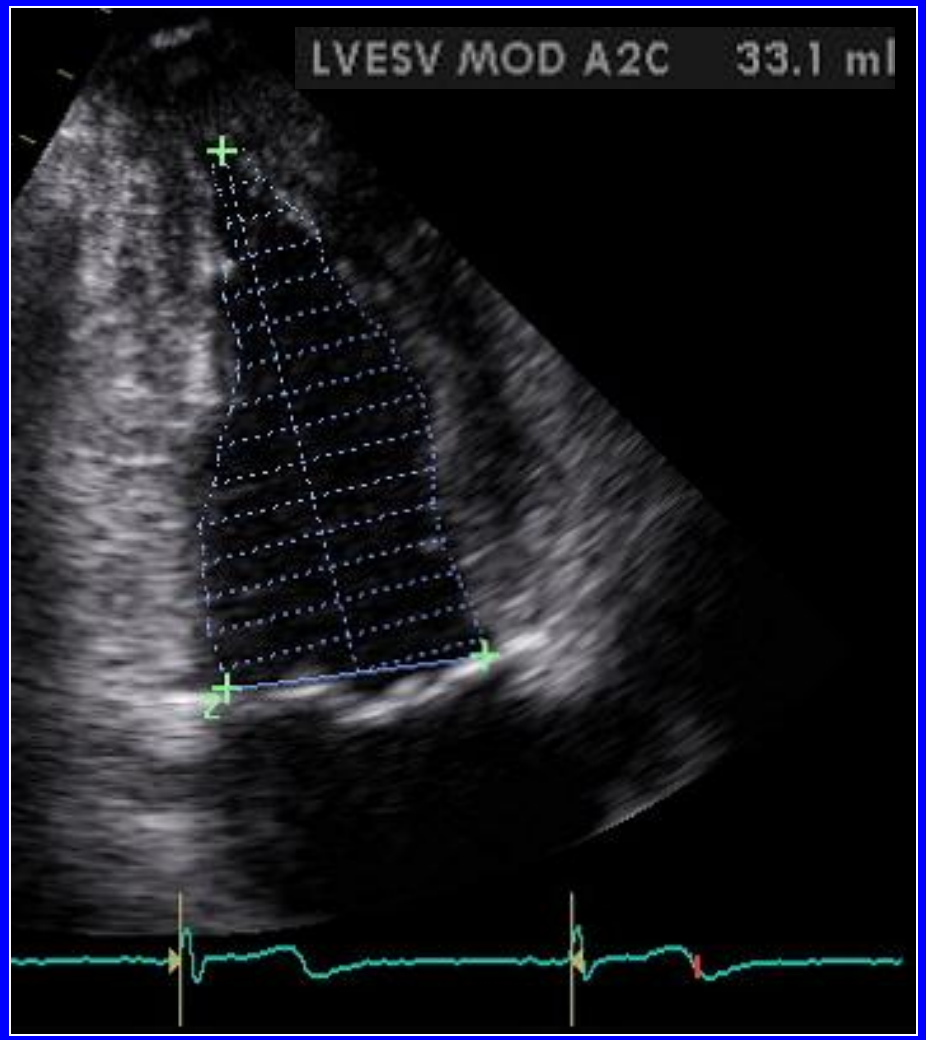
M aged 30, triathlete





SV = 49 ml





EF Biplane	65.3 %
LVEDV MOD BP	117.3 ml
LVESV MOD BP	40.7 ml

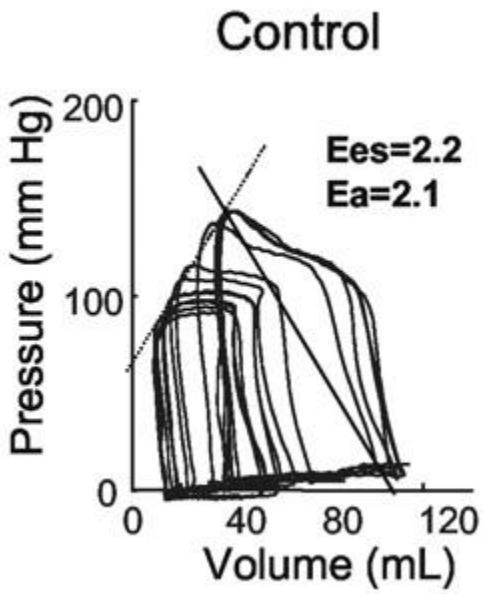
VAC = ESV / SV

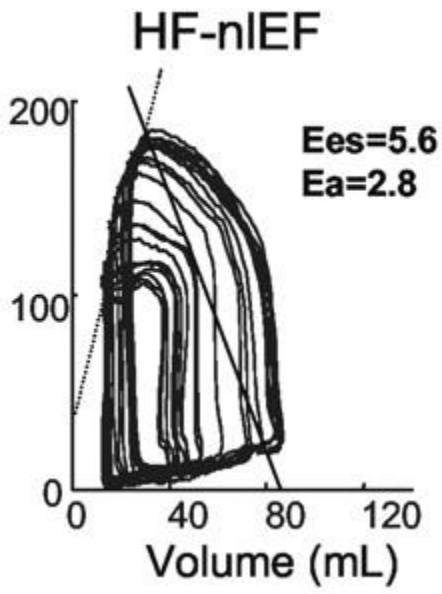
= 41 / 49 = 0.84

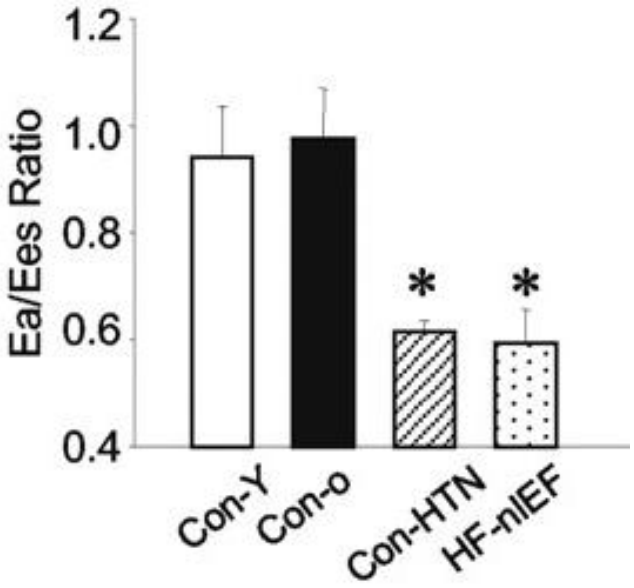
Combined Ventricular Systolic and Arterial Stiffening in Patients With Heart Failure and Preserved Ejection Fraction

Implications for Systolic and Diastolic Reserve Limitations

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







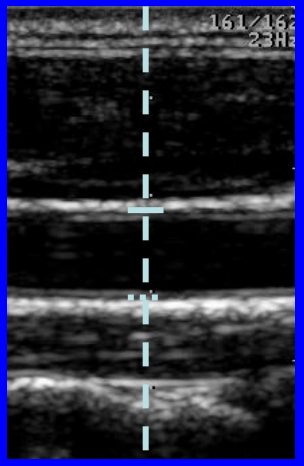
Circulation 2003; 107: 714-20

also – Margulescu A et al, Am J Cardiol 2012 (e-pub)

Imaging arterial stiffness

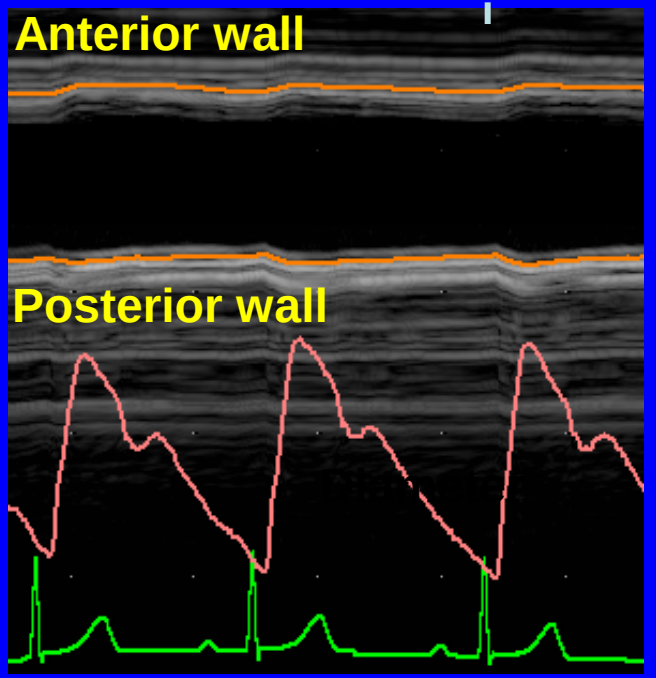
- **BETA INDEX**
  - Stiffness
  - Adjusted for BP
  - No units

$$\beta = \frac{\log_n (Ps / Pd)}{(Ds - Dd / Dd)}$$



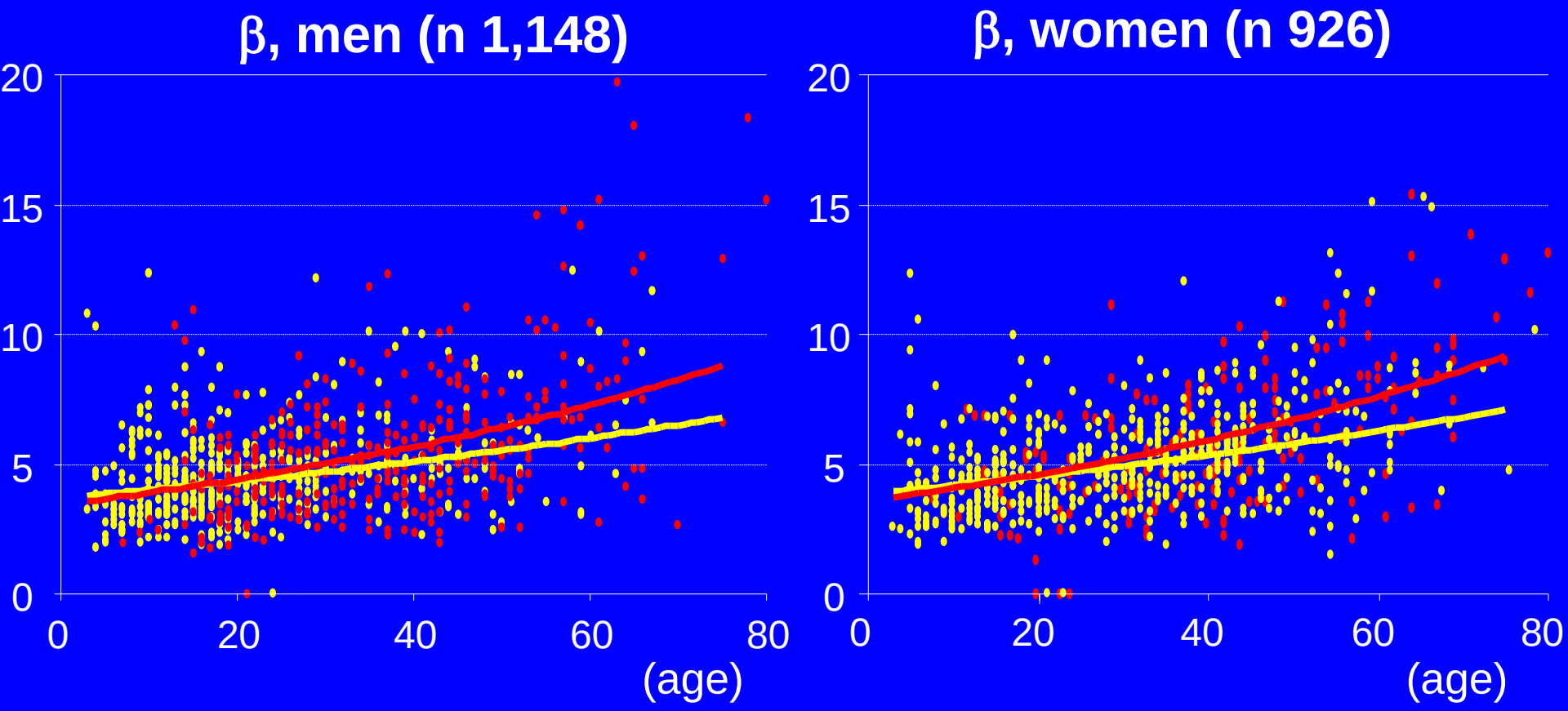
- **EPSILON**
  - Peterson's pressure-strain elastic modulus
  - Measured in kPa

$$Ep = \frac{(Ps - Pd)\, Dd}{(Ds - Dd)}$$



Normative values of conduit arterial function

Influence of gender, age, and body mass index



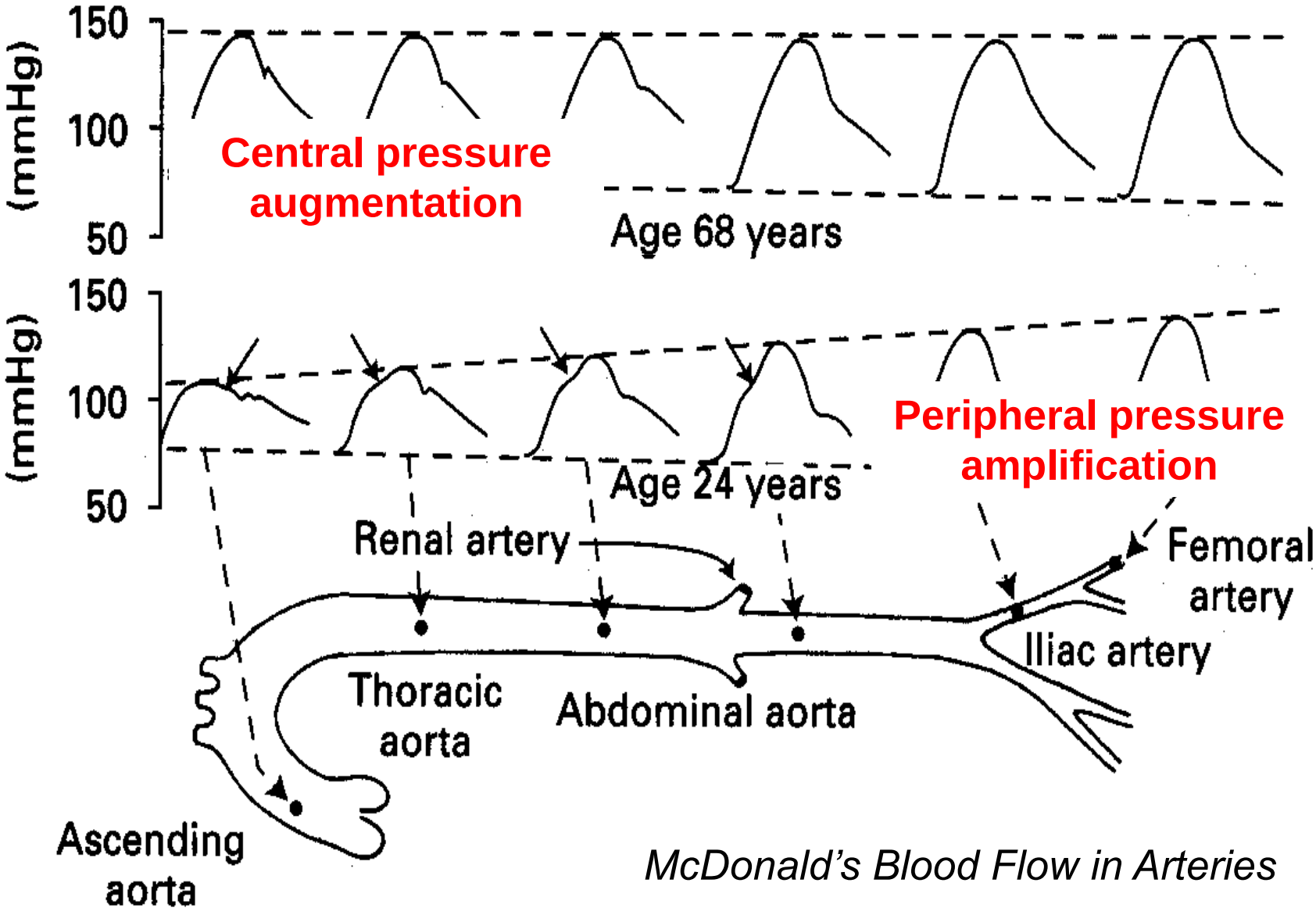
ETIC E-Tracking International Collaboration

2,074 healthy subjects aged 3-85 years

Normal weight

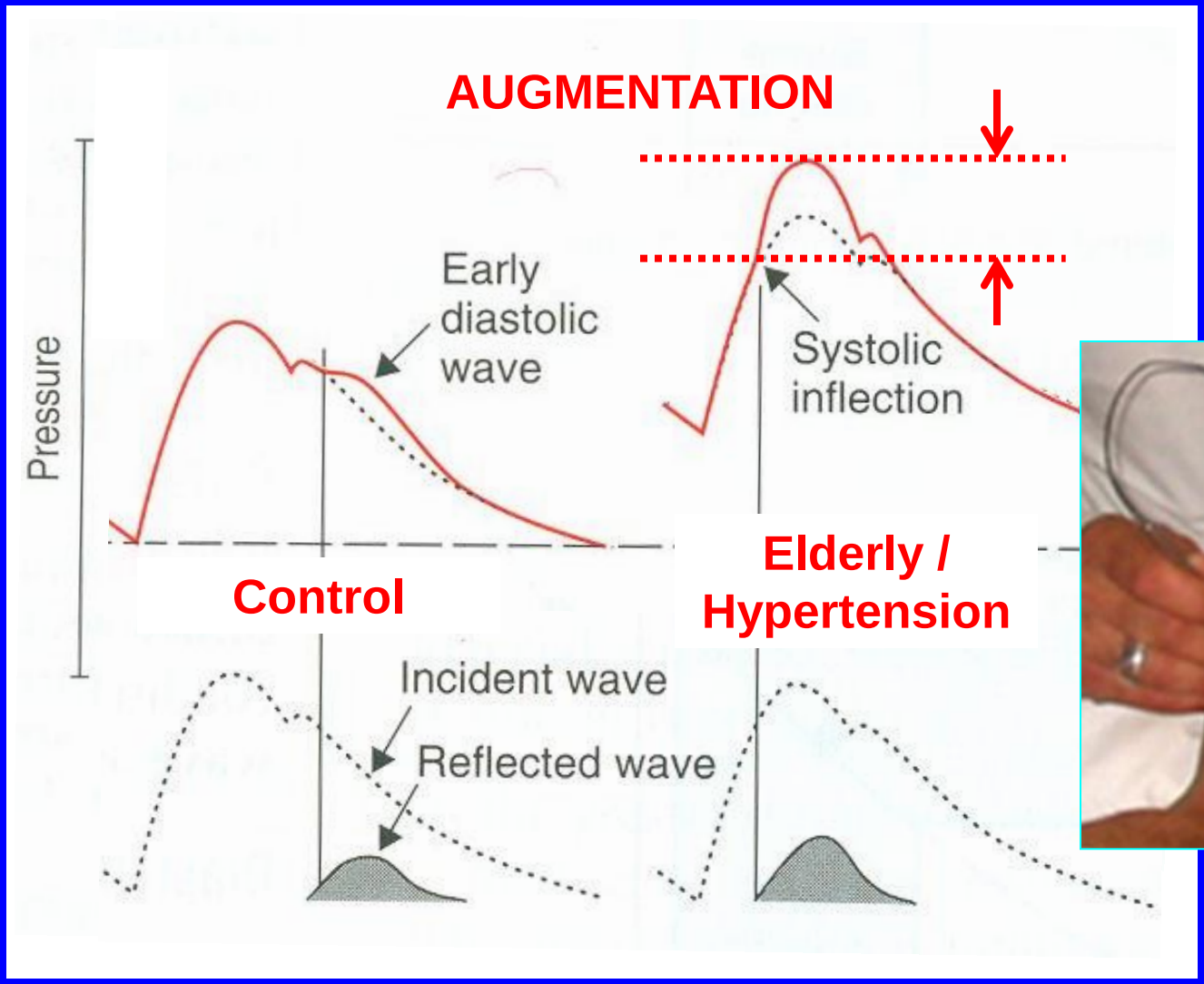
Overweight

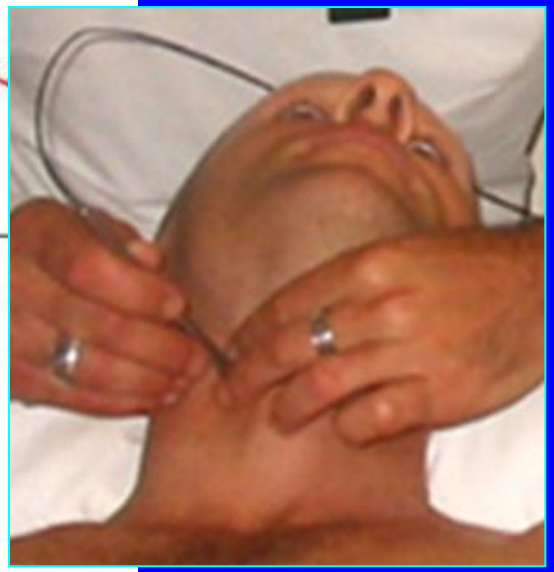
Central arterial pressure changes with age



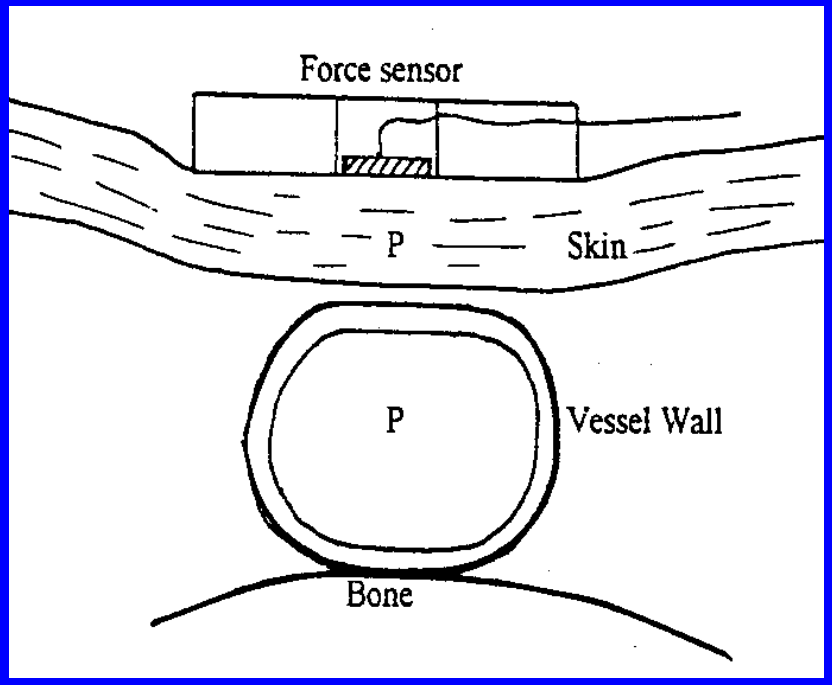
McDonald's Blood Flow in Arteries

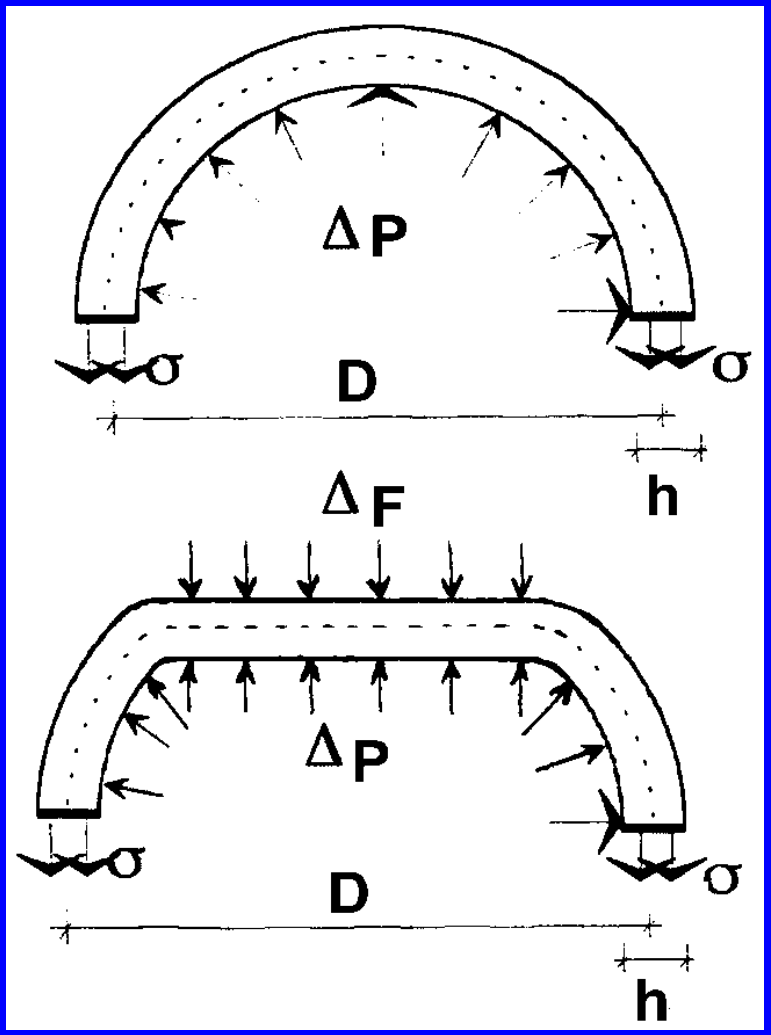
“Direct” estimation of central arterial pressure





Principles of aplanation tonometry



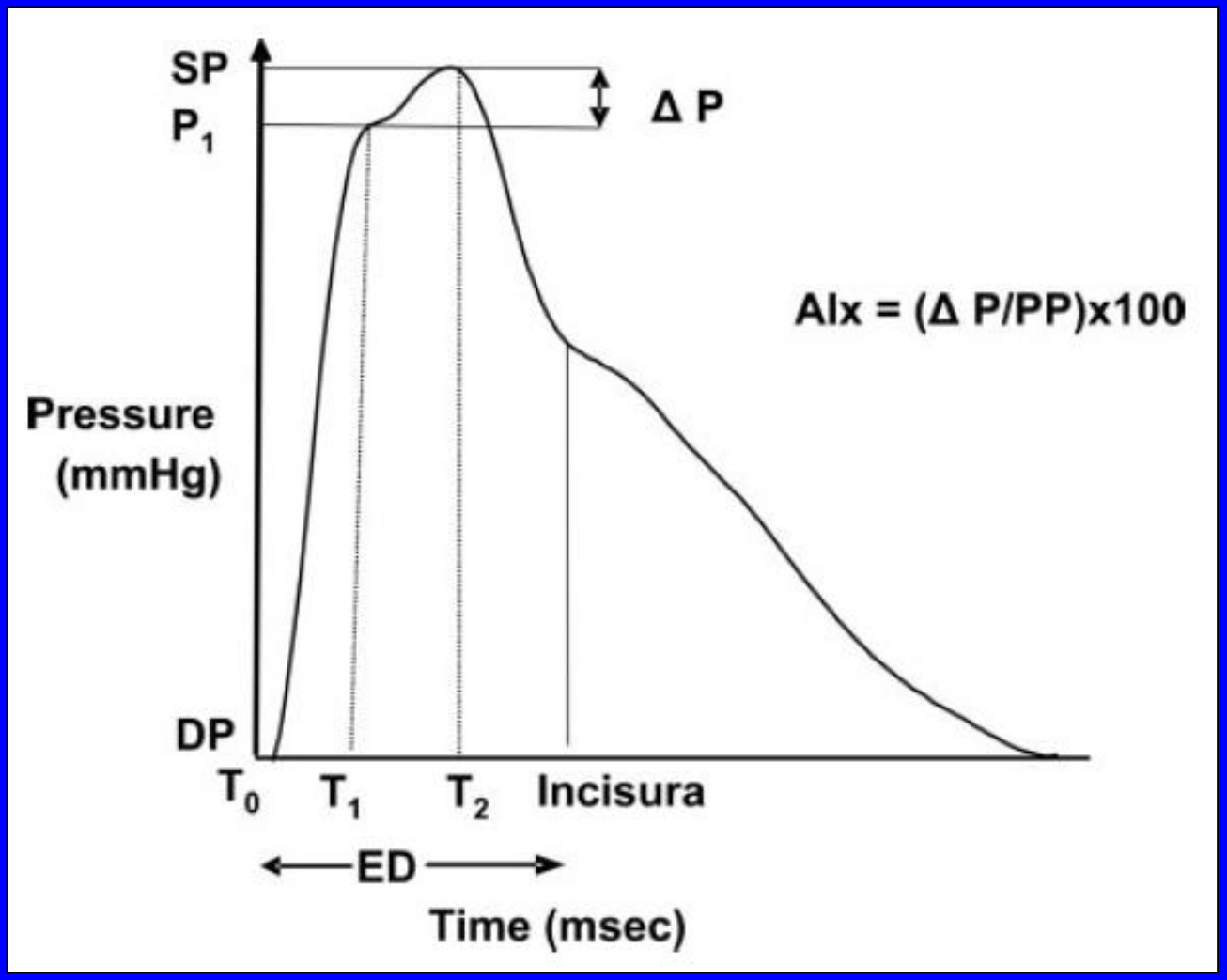


Diameter is scaled to systolic and diastolic pressure

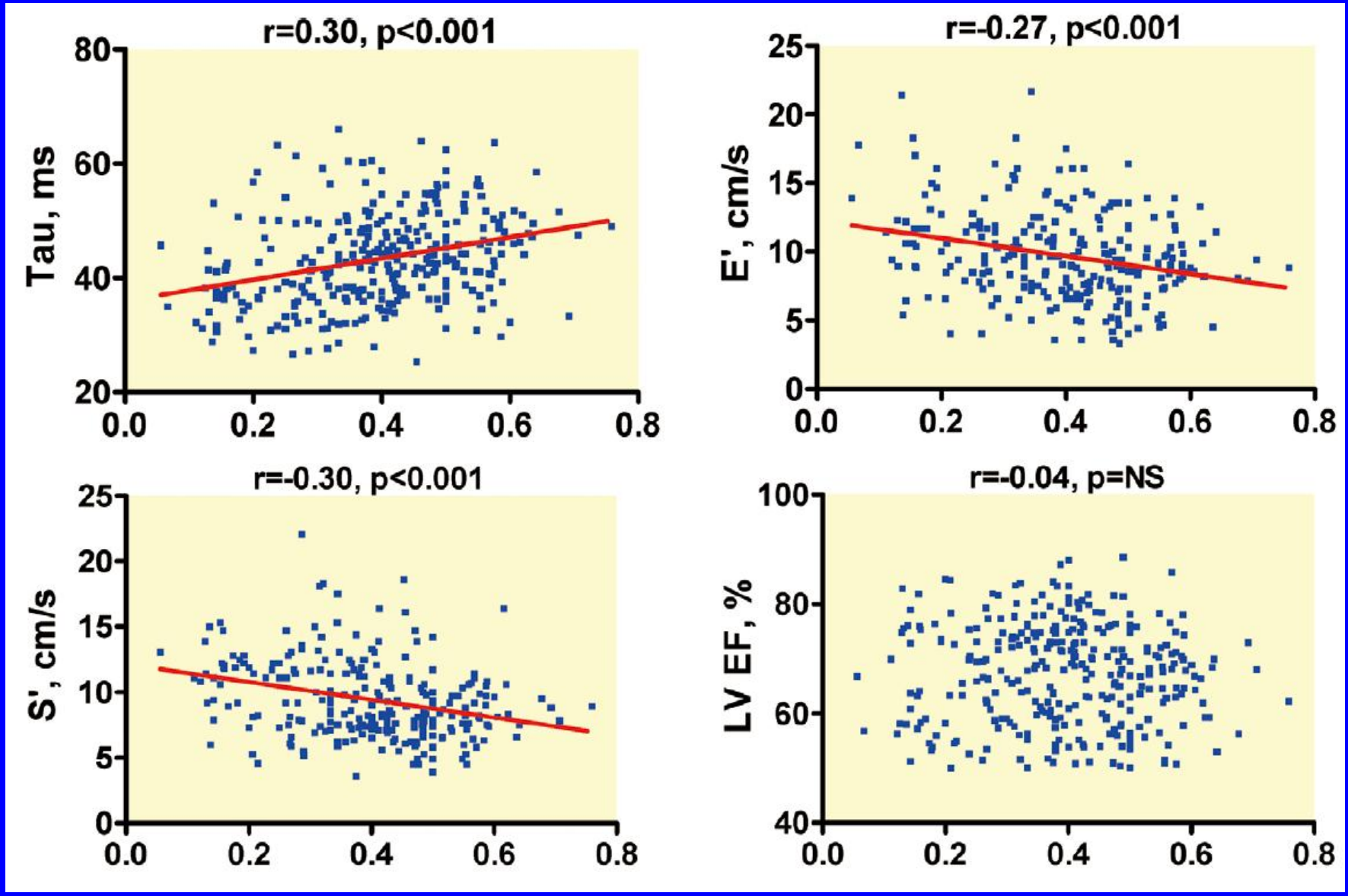
Assumed that diameter and pressure are linearly related

Aplanation tonometry of a peripheral artery (radial)

Pulse wave analysis with “transfer function”

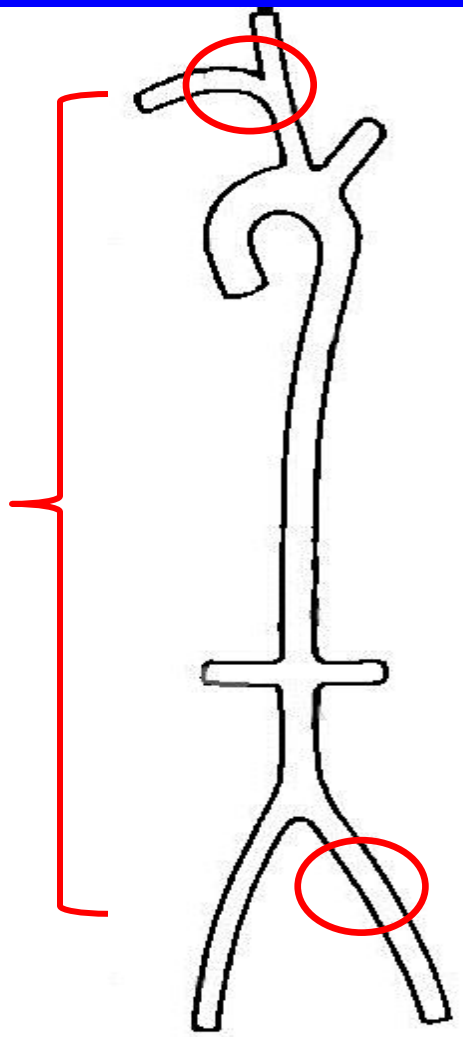


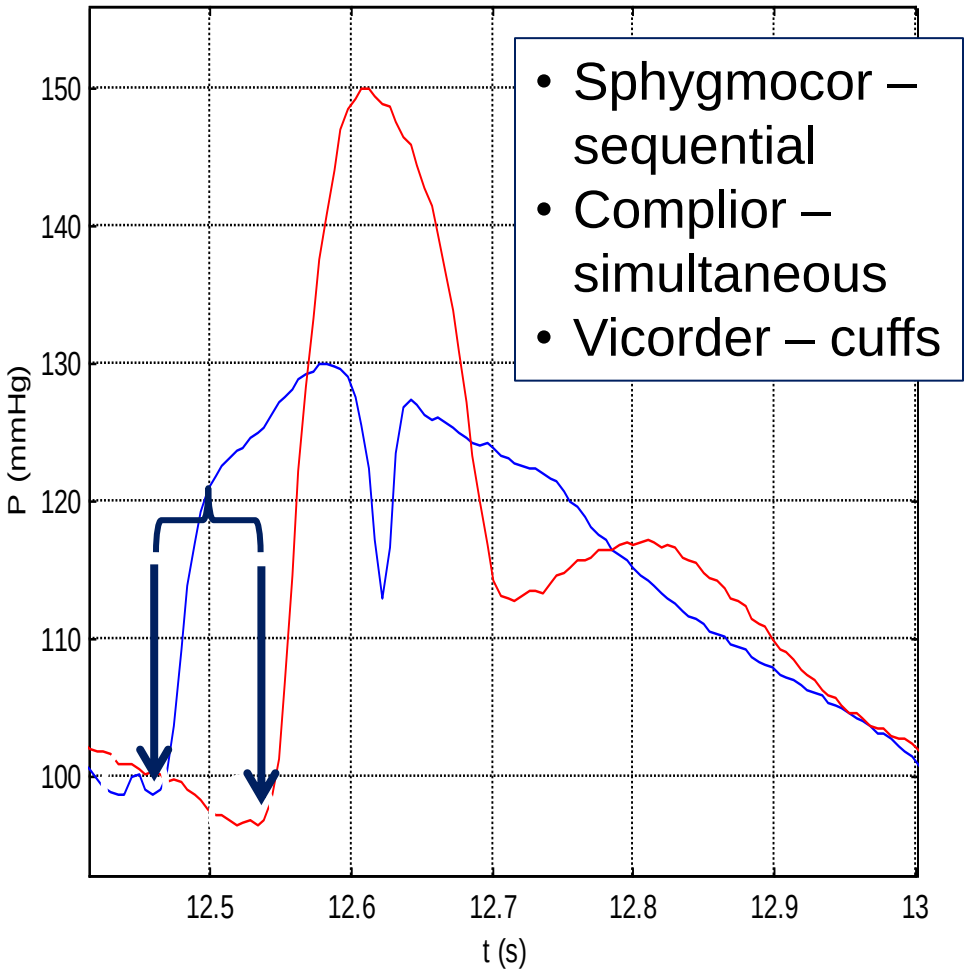
Impact of arterial load (AI) on LV function (n=303)



Fukuta H et al, Circ J 2010; 74: 1900-5

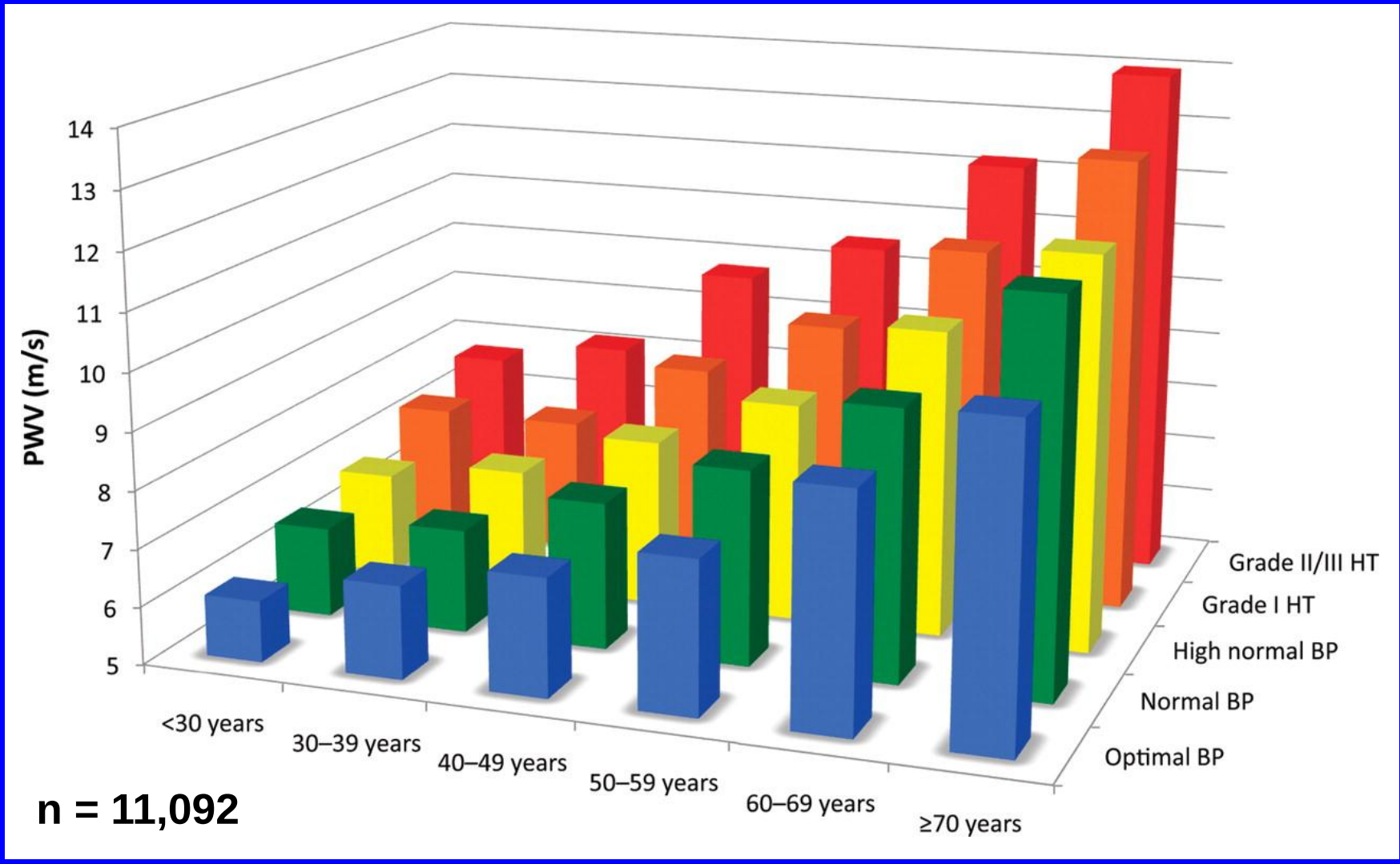
Non-invasive estimation of pulse wave velocity





PWV = transit time ÷ distance (brachial – femoral)

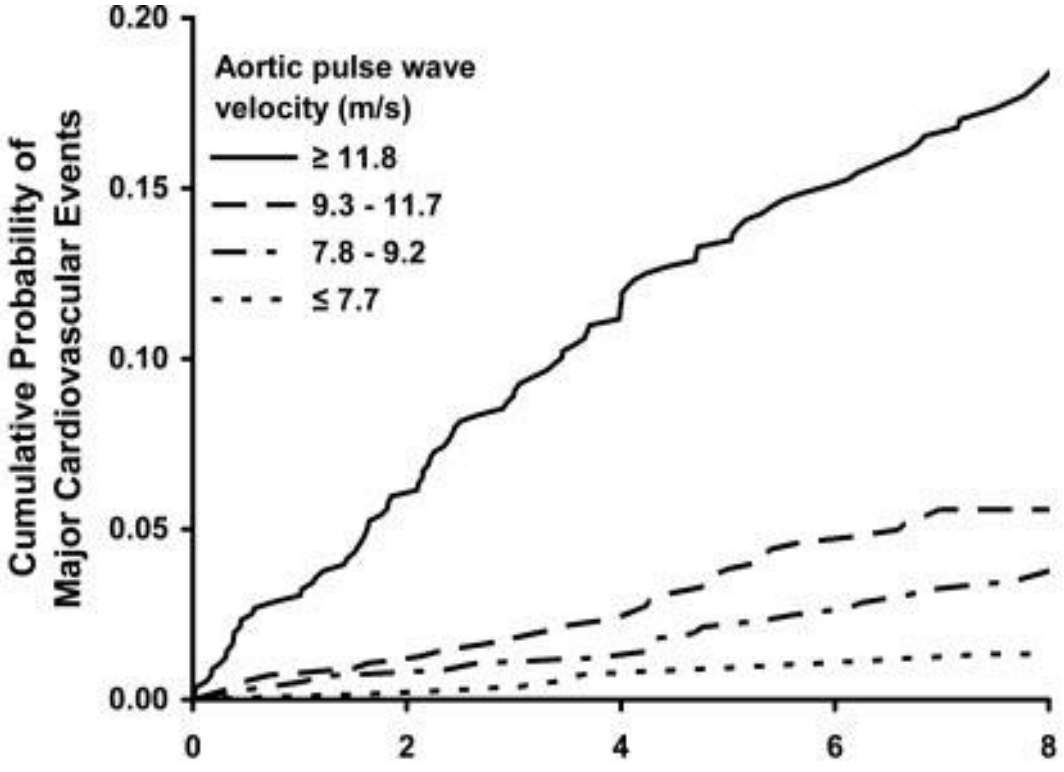
Reference values for pulse wave velocity



Eur Heart J 2010;31:2338-50

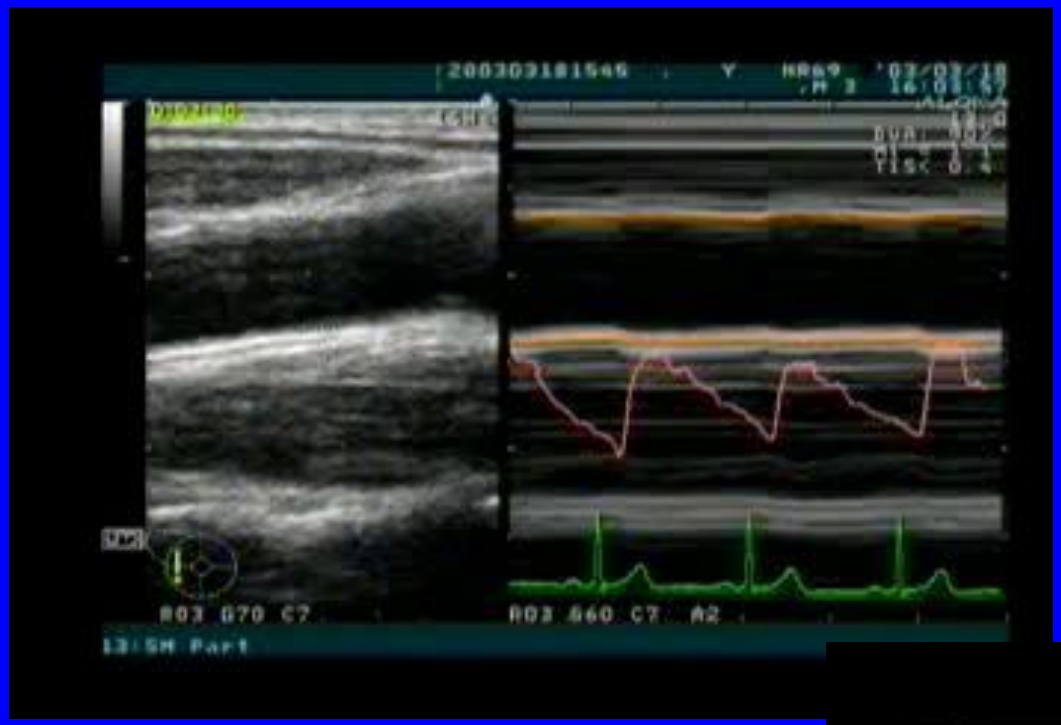
Prognostic utility of aortic pulse wave velocity

PWV added independent prognostic value to the standard Framingham risk score



Number at risk: Aortic pulse wave velocity (m/s)					
≥ 11.8	560	513	462	424	161
9.3 - 11.7	555	542	529	502	246
7.8 - 9.2	573	561	551	537	278
≤ 7.7	544	541	535	531	275

Mitchell GF et al, Circulation 2010; 1221: 505-11

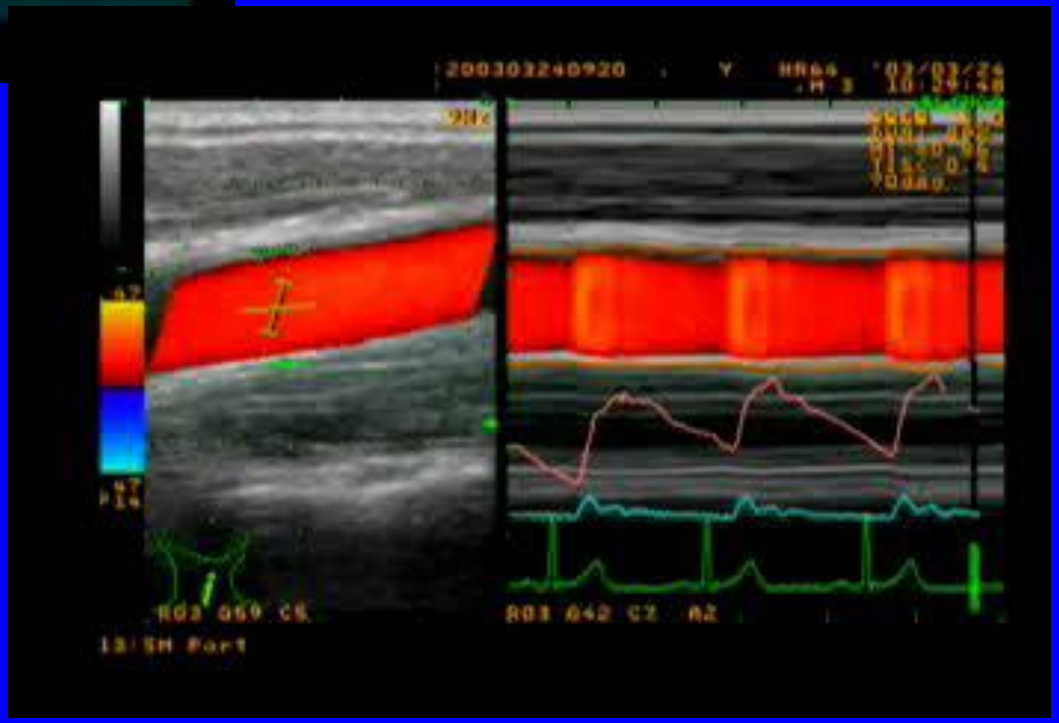


Wall tracking of common carotid artery

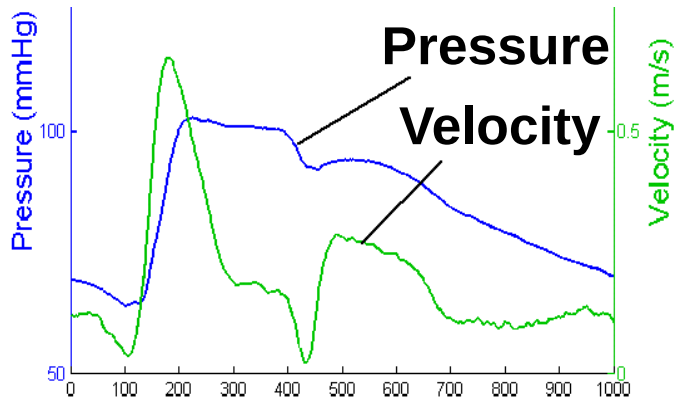
Diameter / distension waveform, calibrated as estimate of pressure

Simultaneous measurement of blood velocity at same site

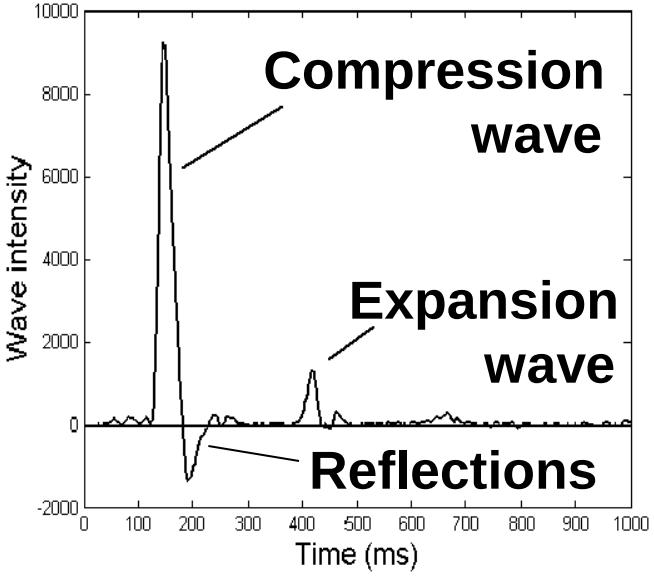
→ **Non-invasive wave intensity**



Arterial wave intensity



dP/dt.dU/dt

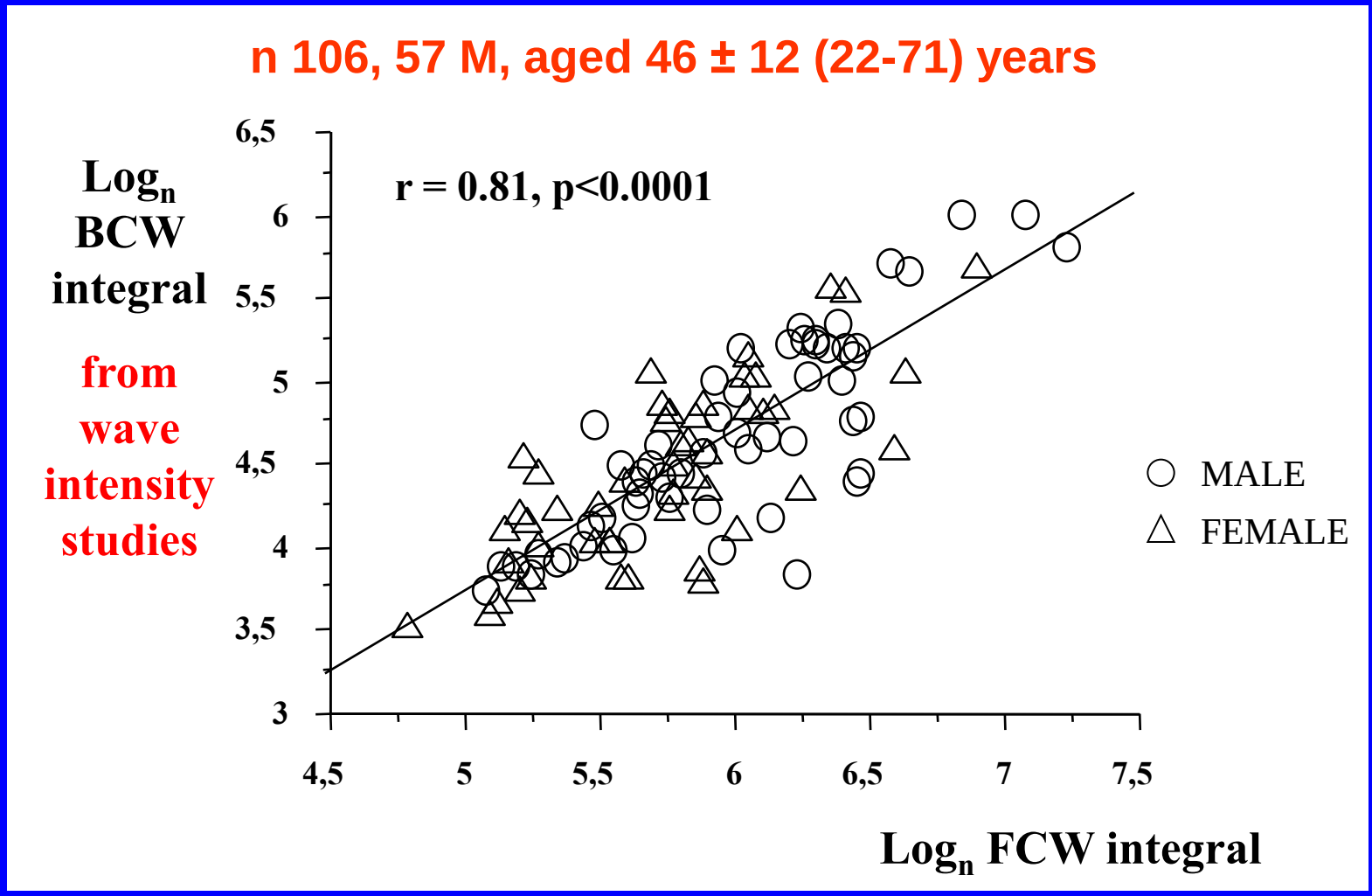


There are 4 types of waves =

	U	P
Forward compression	↑	↑
Forward expansion	↓	↓
Backward compression	↓	↑
Backward expansion	↑	↓

The time integral of wave intensity is energy

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

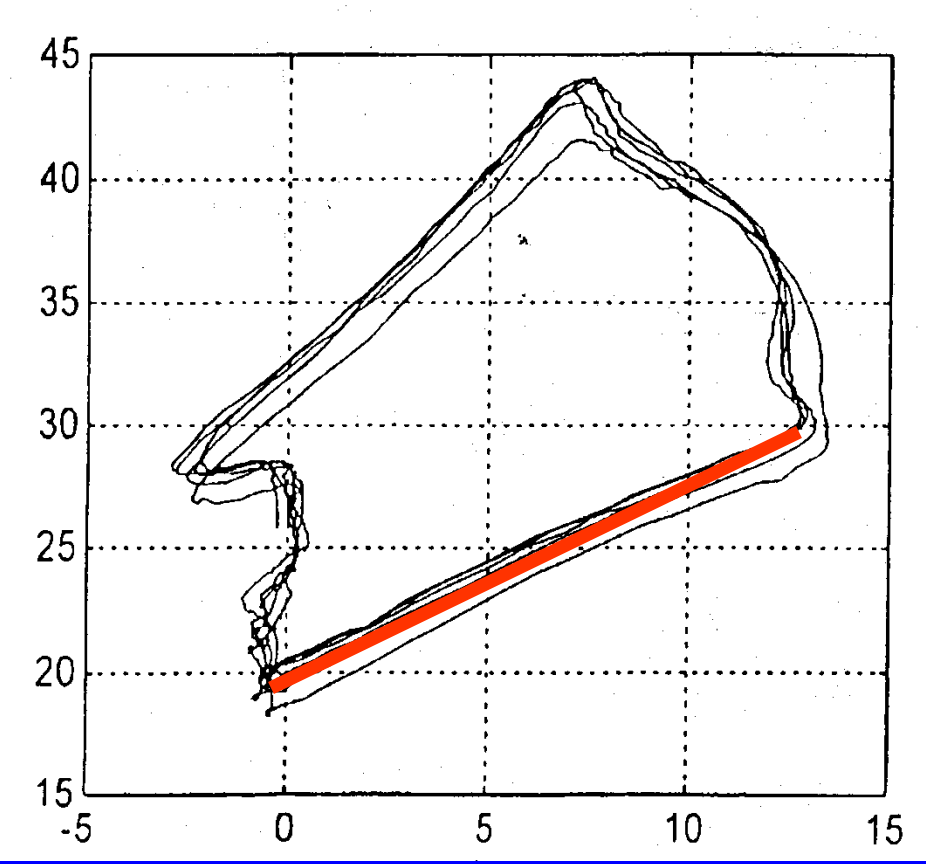


Rakebrandt F et al, Ultrasound Med Biol 2009; 35: 266-77

Calculation of wave speed

Early systolic slope of pressure / velocity loop

Pressure



Velocity

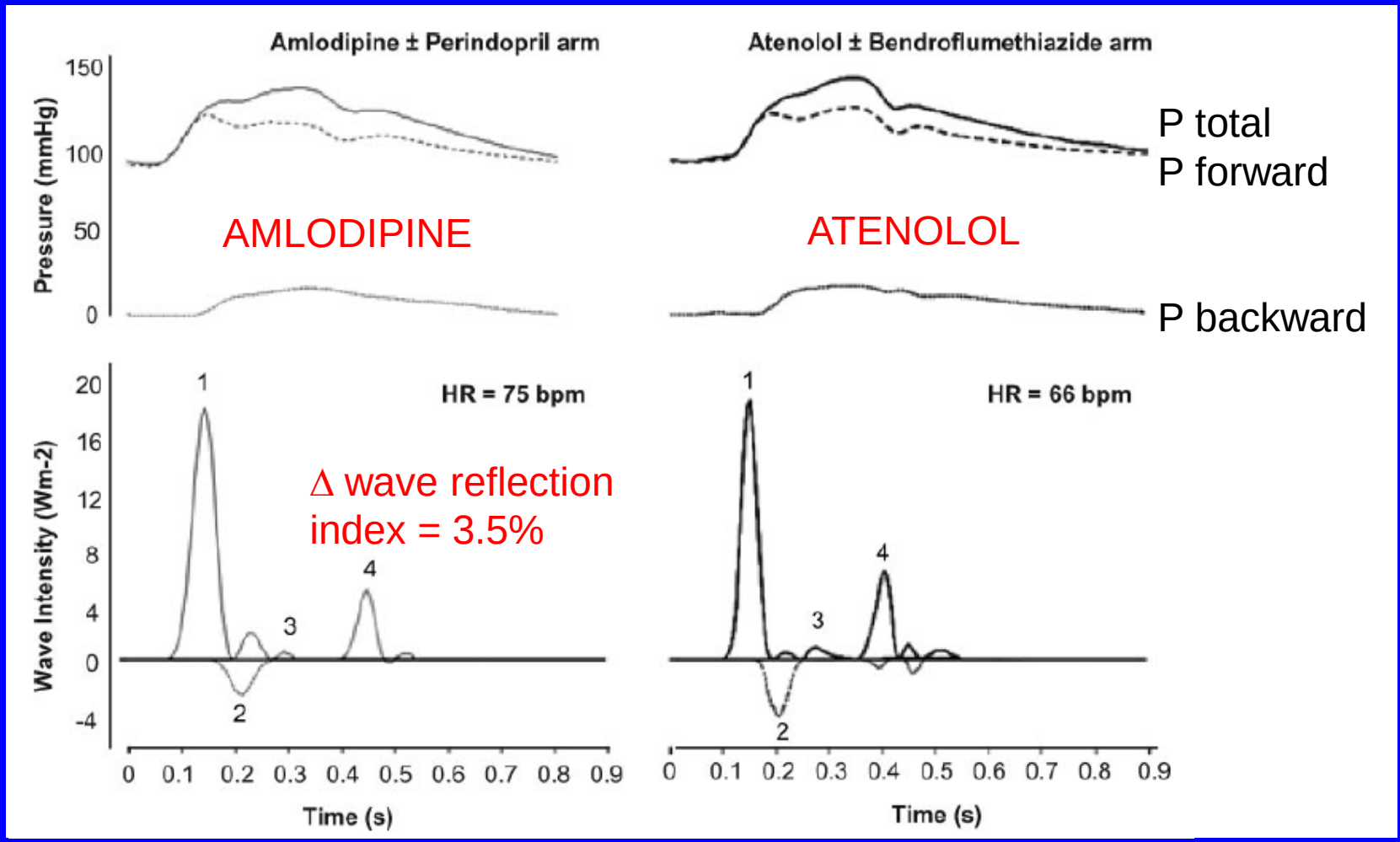
Wave speed

$c = (dP/dU)/\rho$

Harada A et al, Heart Vessels 2002; 17: 61

Comparison of antihypertensive drugs (ASCOT)

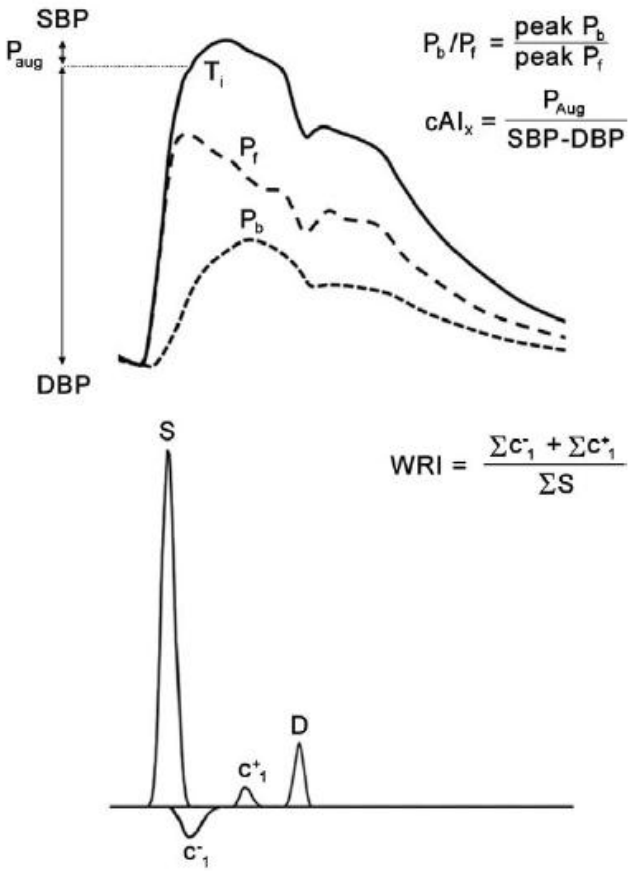
Difference in the magnitude of mid-systolic reflections



Manisty C et al, Hypertension 2009; 54: 724-30

Wave Reflection Predicts Cardiovascular Events in Hypertensive Individuals Independent of Blood Pressure and Other Cardiovascular Risk Factors

An ASCOT (Anglo-Scandinavian Cardiac Outcome Trial) Substudy



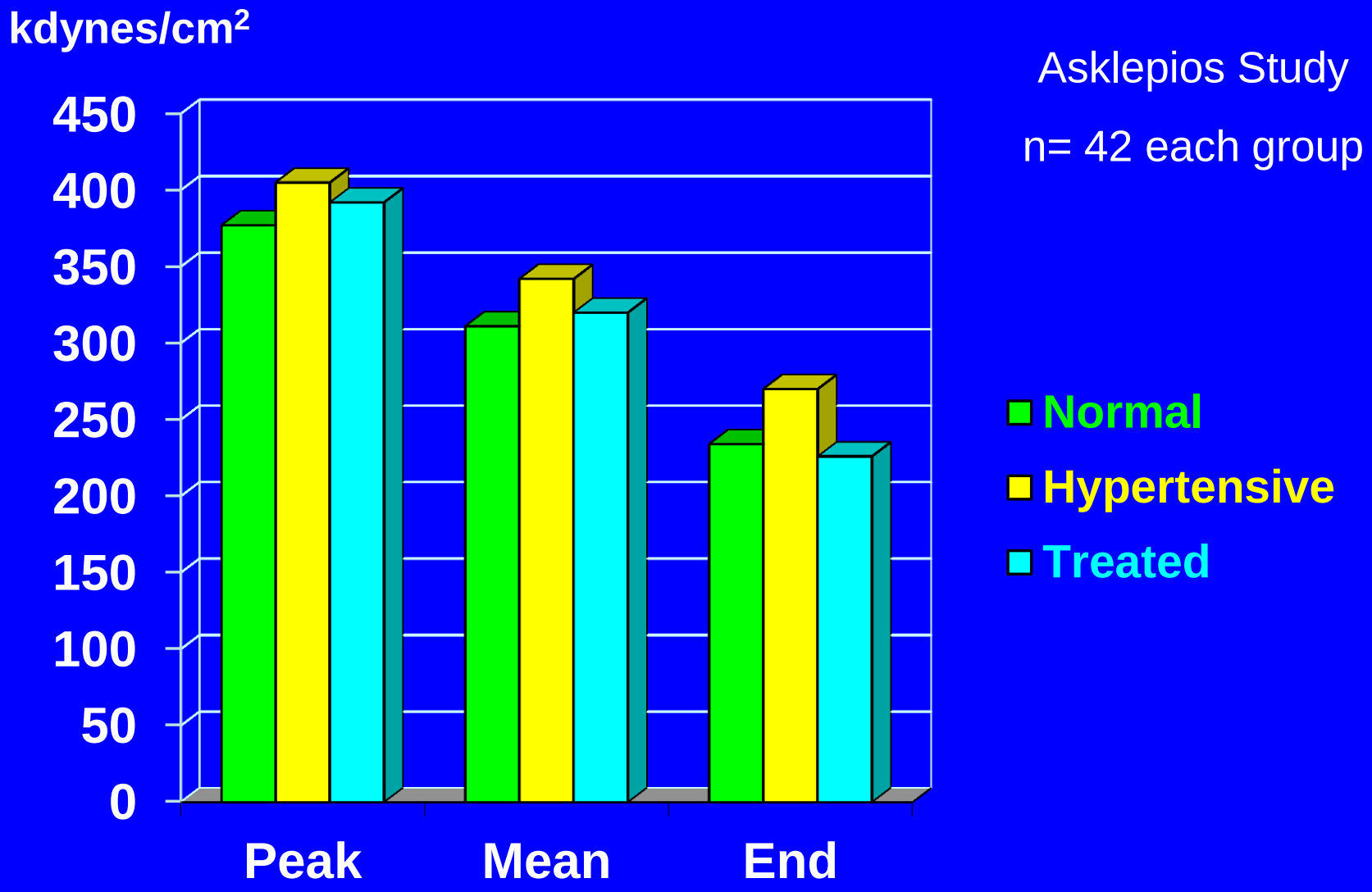
259 subjects followed for mean of 5.9 years

Multivariate model	Predictors of Cardiovascular Events		
Log WRI	2.17	(1.08–4.37)	0.03
DBP, mm Hg	0.94	(0.90–0.98)	0.01
Carotid PP, mm Hg	1.01	(0.99–1.03)	0.2

	Beta Coefficient	SE	p Value
Model to predict LVMI			
Log WRI*	6.6	3.2	0.04
Log WRI† (fully adjusted)	7.3	3.3	0.03
P_b/P_f*	10.0	8.9	0.3
P_b/P_f† (fully adjusted)	14.7	10.7	0.2
cAI_x*	0.1	0.1	0.4
cAI_x† (fully adjusted)	0.2	0.2	0.2
Carotid wavespeed*	0.8	0.5	0.1
Carotid wavespeed† (fully adjusted)	0.7	0.5	0.2

Manisty C et al, JACC 2010; 56: 24-30

Circumferential myocardial stress during systole



Chirinos JA et al, Circulation 2009; 119: 2798-2807

Analysis of treatment of hypertension

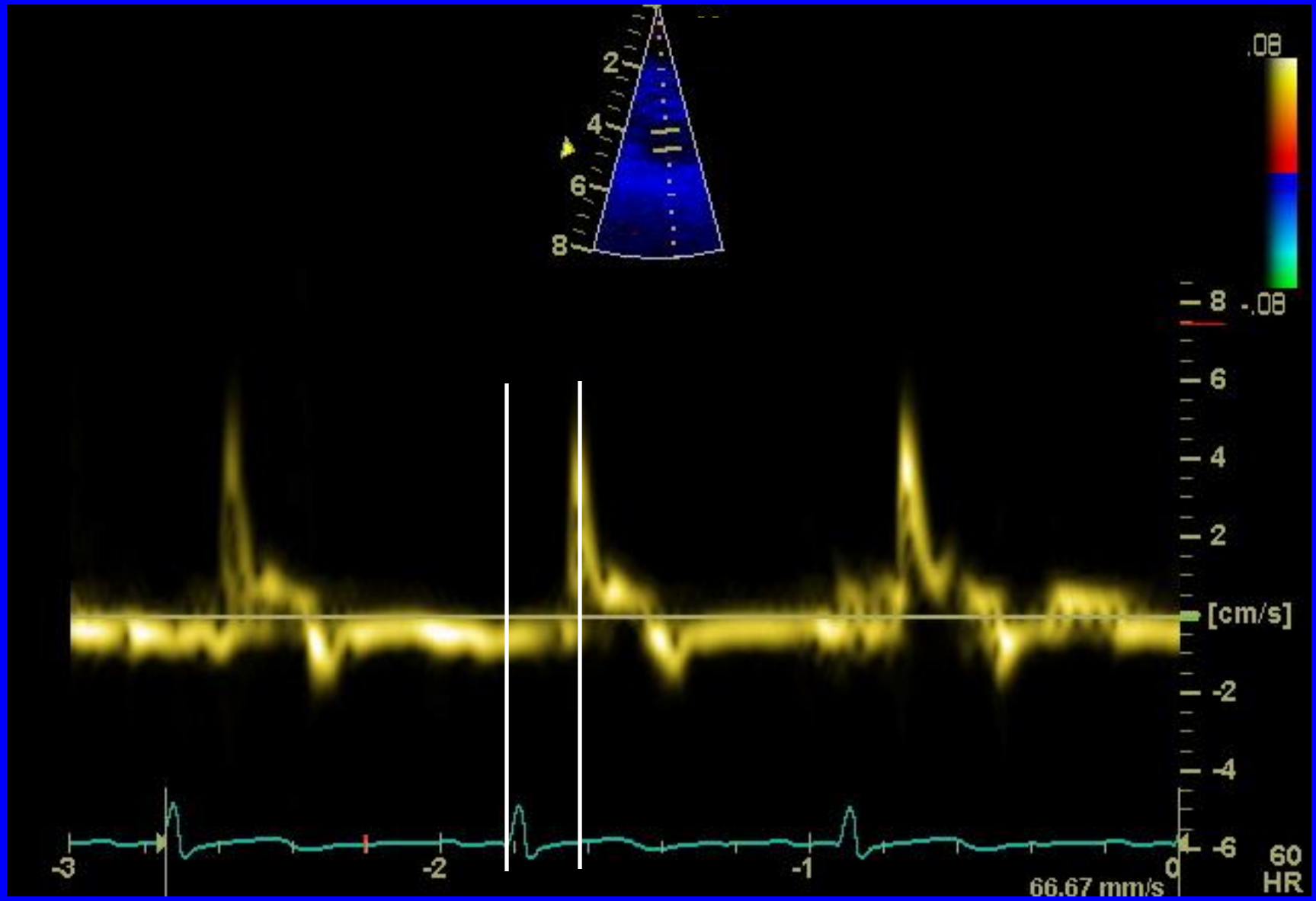
Prospective open-label study of barnidipine

	20 well controls	21 untreated hypertensives	After 6 m treatment
Blood pressure	126 / 78	159 / 96***	138 / 81***
Augmentation index	13 ± 5	22 ± 7**	17 ± 8**
Beta index	10 ± 3	11 ± 3	10 ± 3
Epsilon	126 ± 41	175 ± 49**	155 ± 53**
Arterial compliance	0.9 ± 0.3	0.6 ± 0.2**	0.7 ± 0.2
Wave speed	6.8 ± 1.0	7.8 ± 1.0**	7.5 ± 1.7
Forward pressure	108 ± 7	137 ± 17***	124 ± 14*
Backward pressure	17 ± 5	21 ± 6*	18 ± 3

** <0.05*
*** <0.01*
**** <0.001*

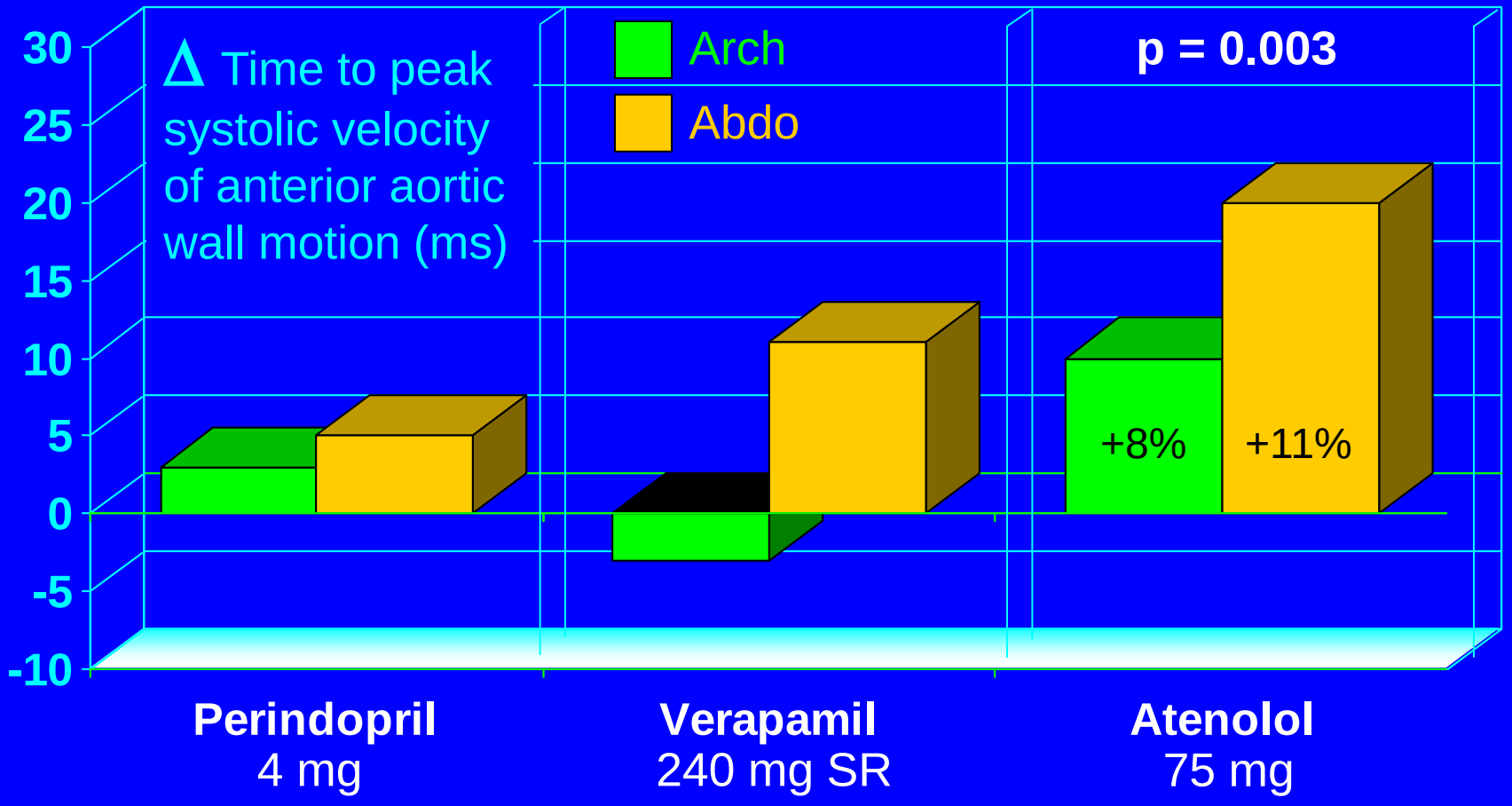
Palombo C, Clin Ther 2009;31:2873-85

Tissue velocity of aortic wall motion



Aortic wall motion in Marfan syndrome

Prospective, double-blind, randomised cross-over



Atenolol also reduced heart rate by 16%
– so it reduced pulsatile stress

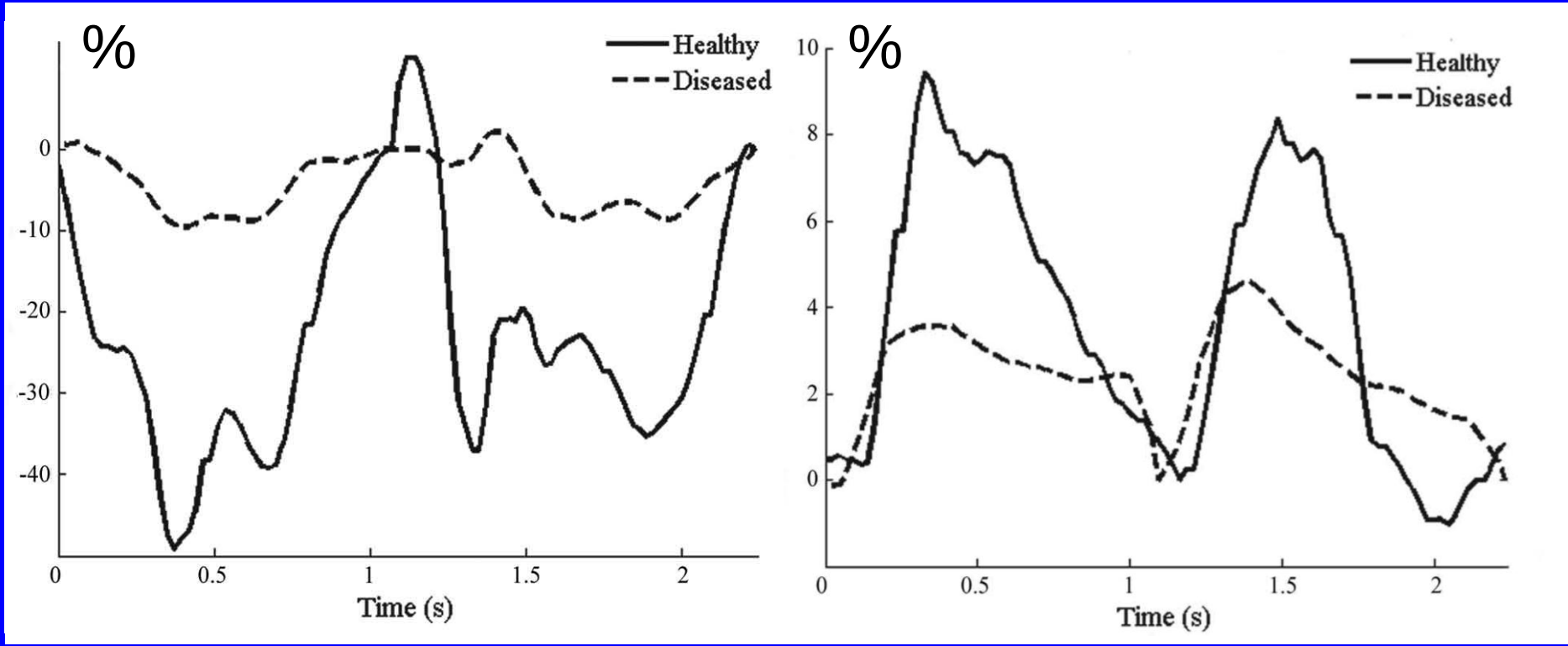
Williams A et al,
Eur J Clin Invest 2012 (e-pub)

Mechanical deformation of conduit arterial wall

Speckle tracking – *" Wave Intensity Wall Analysis "*

Radial strain

Longitudinal strain



Larsson M et al, IEEE Trans Ultrason 2011; 58: 2244-51

also – Bjällmark A et al, Heart Vessels 2011; 26:289-97

Ventriculo-arterial coupling – clinical applications

- Pathophysiological insights from clinical research
  - Mechanisms of ISH, and of LVF in HFNEF
  - Non-uniformity of conduit arterial function
- More accurate diagnosis, better prognosis
  - Pulse wave velocity
  - Mid-systolic wave reflections
- Selecting and monitoring drug treatment
  - to reduce central arterial pressure
  - to delay and reduce mid-systolic reflections
  - to reduce net and pulsatile stress in aorta