Effect of loading and geometry on functional parameters

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Functional Parameters ?

What is function?

The ‘ability’ of the heart to provide sufficient oxygenated blood to the organs (including itself) in all conditions.

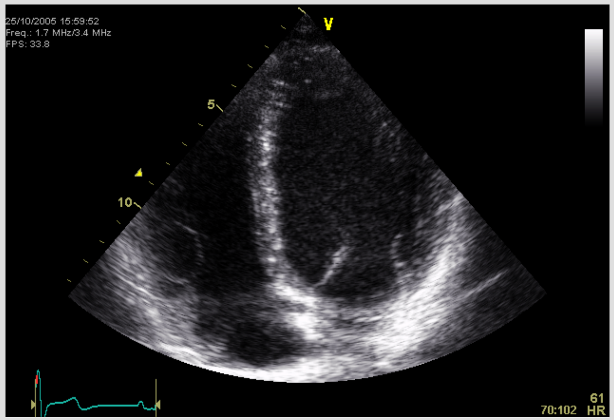
1. Maintain cardiac output at rest and increase at stress
   The heart is a *volume* pump

2. Two circulations with different pressure levels
   Reflected into differential properties of the LV and RV.
   - morphological, functional, ...

3. Cope with changes (acute and chronic) in boundary conditions (load, geometry, size)
   impact on functional parameters

↓

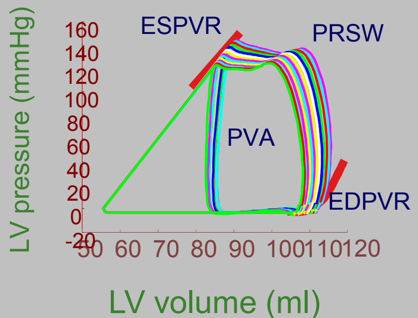
Most *successful* parameters have a ‘load change’ component.



Function: at what level?

Pump performance

Global Function



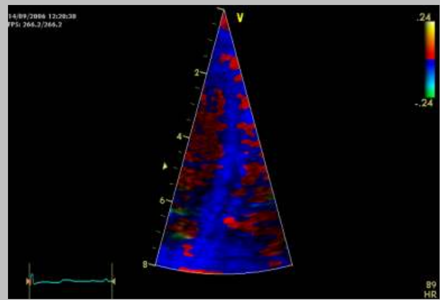
Volumes
Cardiac Output
Ejection Fraction

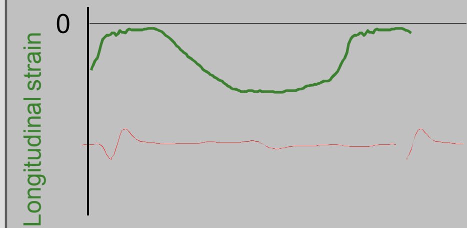
Pressure

Pressure-volume relations

Myocardial function

Regional Function



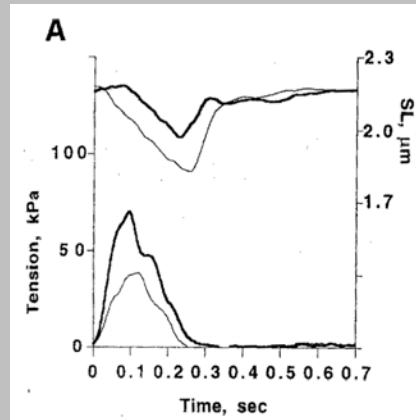


Strain and strain-rate
Myocardial velocities
Annular motion

Wall stress

Intrinsic function

Fiber mechanics

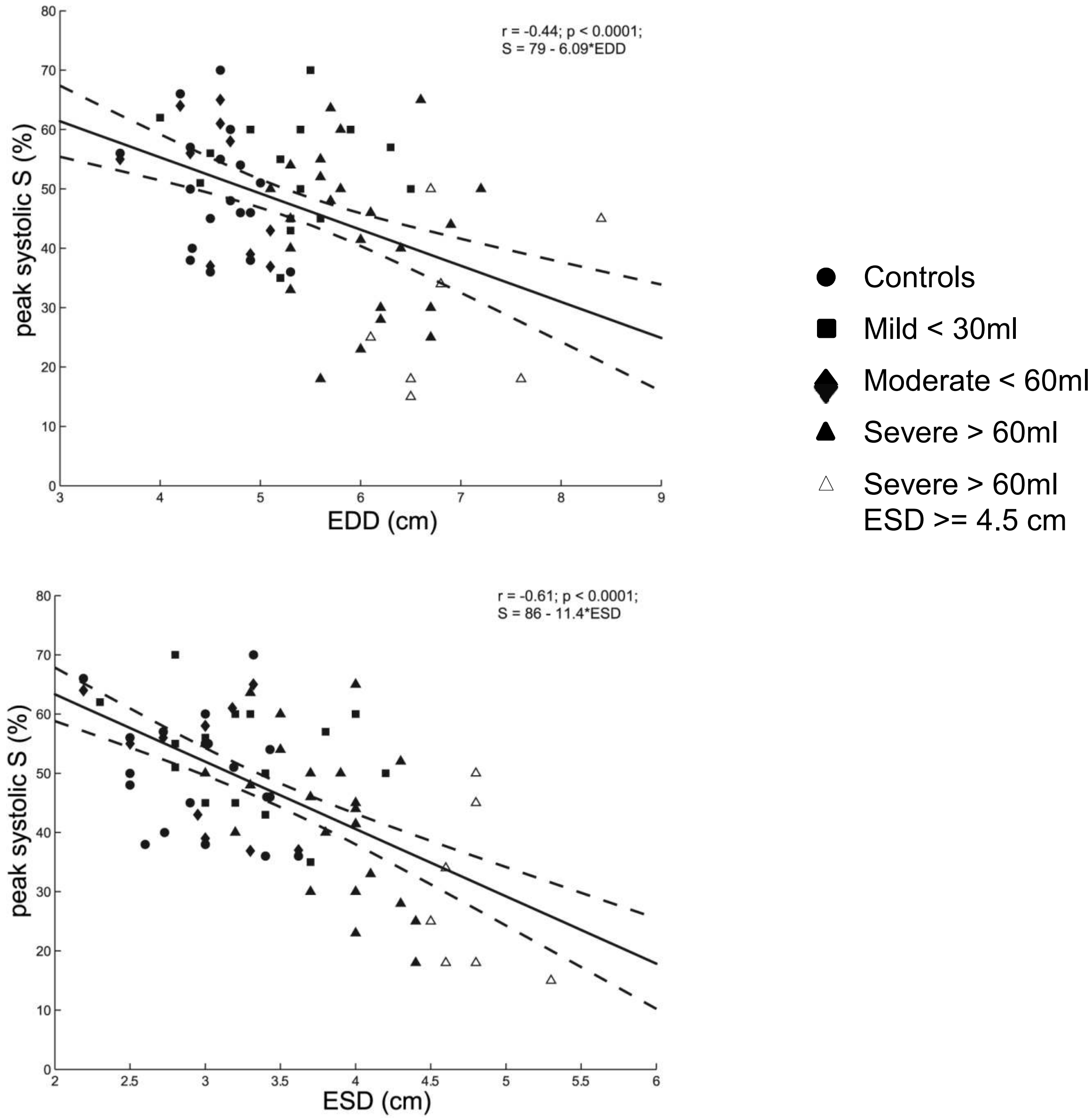


Fiber strain

Fiber stress

understand

Mitral Regurgitation

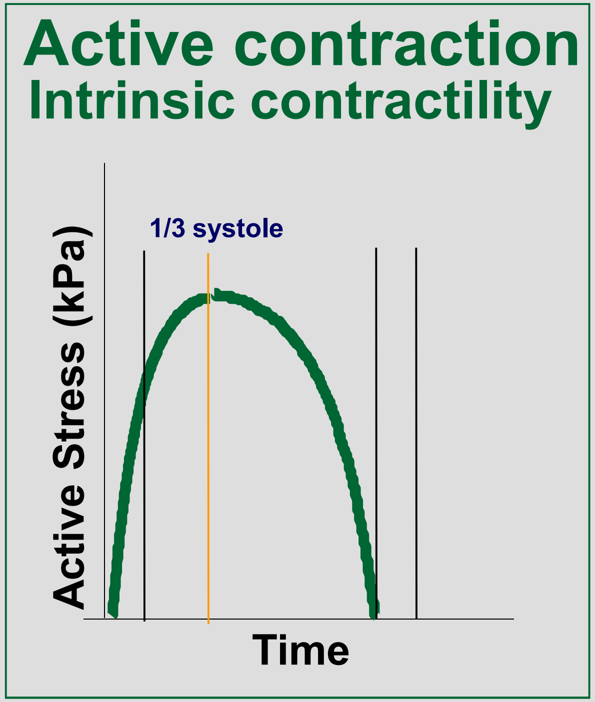


Relation between deformation and size.

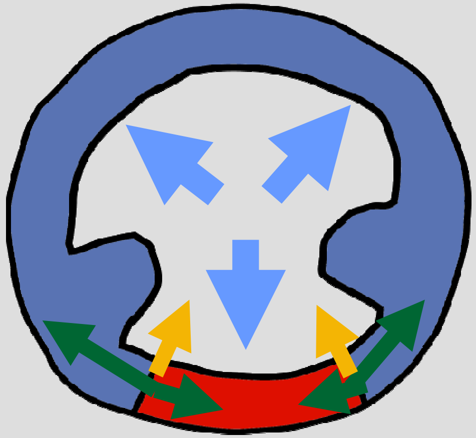
Is strain useful ?

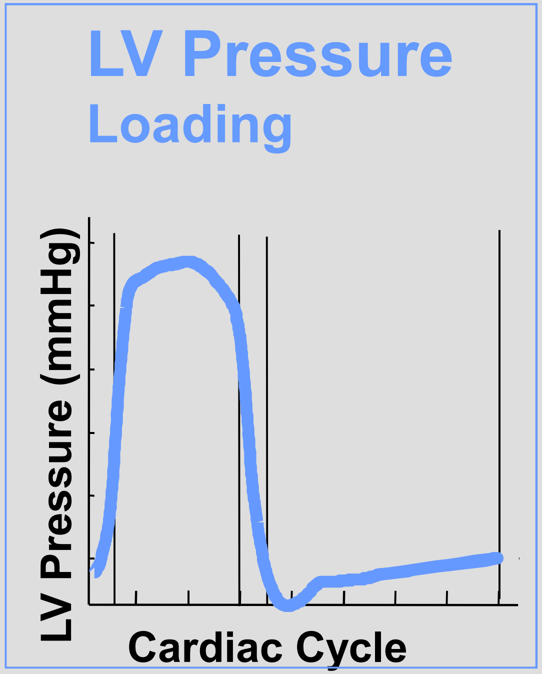
Marciniak A, Claus P,... Eur Heart J. 2008

The ventricle as a mechanical system



Guccione et al. J. Biomech. 30(2) 1997





Measured

'Shear forces'

Interaction of segments with differential thickness

include ←

Elastic forces

non-linear tension/strain relation

Guccione et al. J. Biomech. 28(1) 1994

Predictions in simplified dynamical model

Geometry based on an ellipsoid model.
Conservation of myocardial volume
With extrapolated dynamics from the spring mass model.

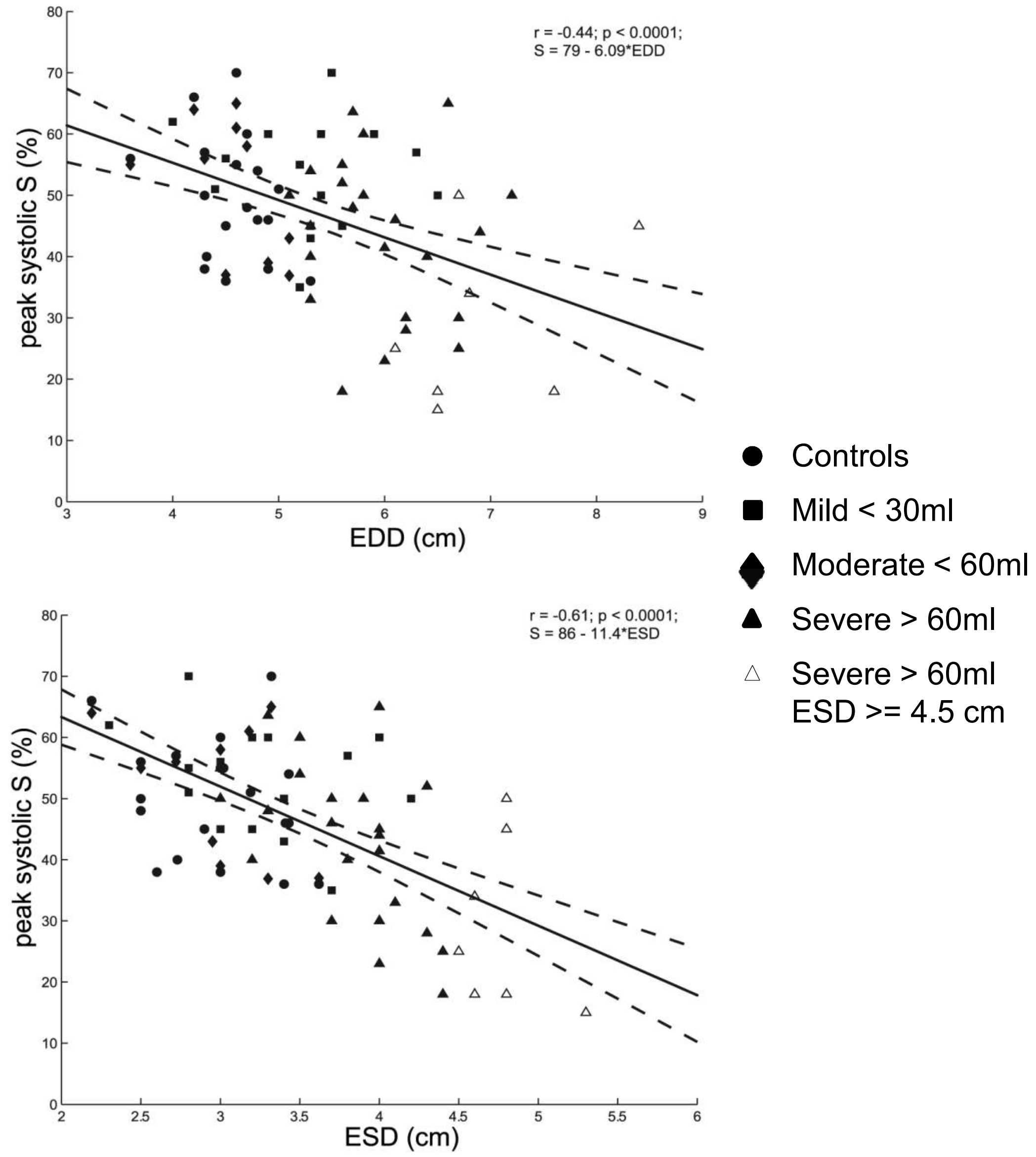
Loading ranges:
Afterload: 120-160 mmHg
Preload: 0-50 mmHg

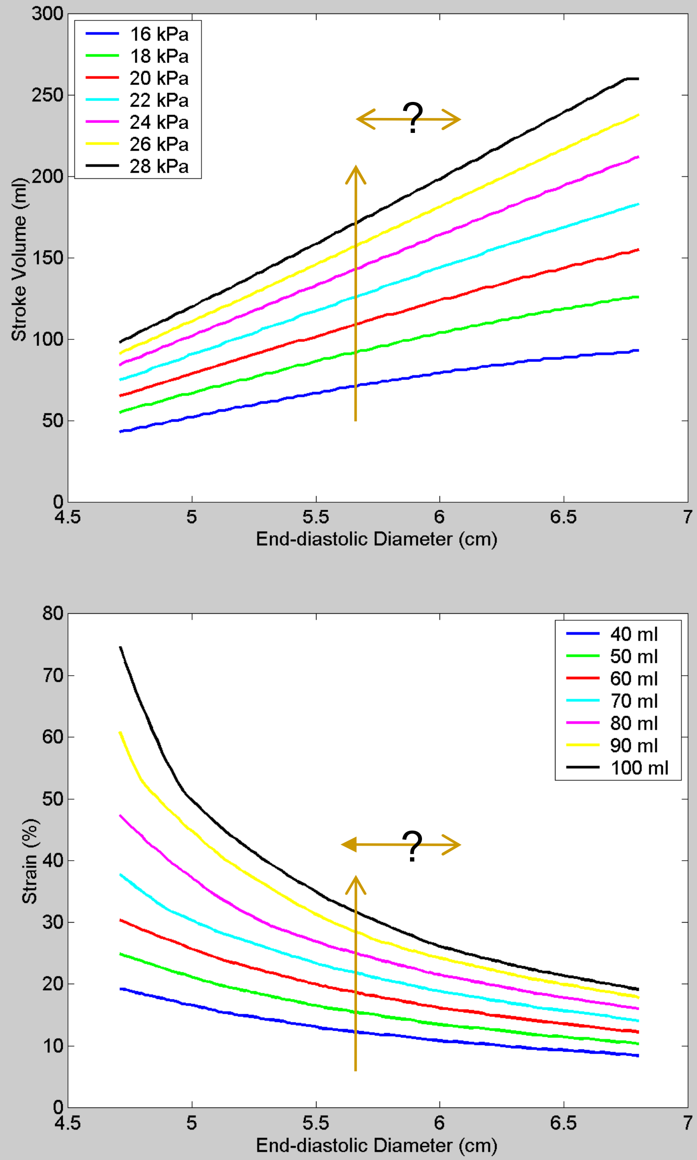
Peak active force range:
30 – 70 kPa

Assumptions

No changes in elasticity
No regional differences

Strain versus size in volume overload

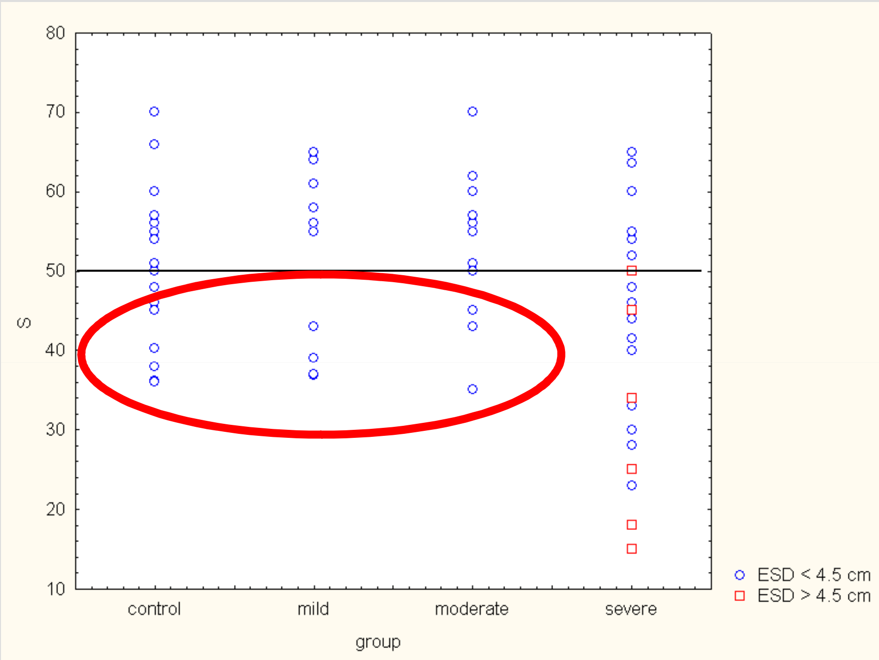




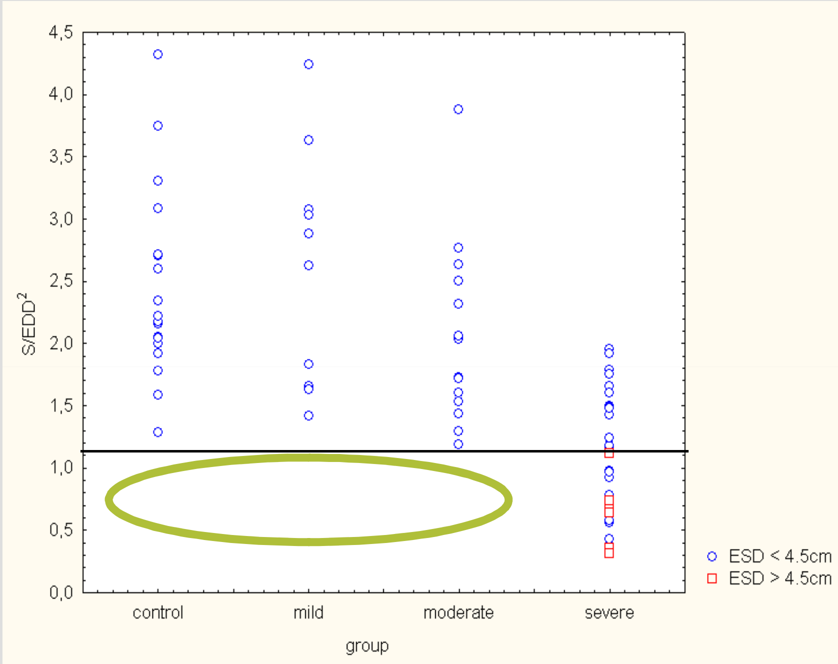
Marciniak A, Claus P,... Eur Heart J. 2008

Correction for ventricular size ?

End-systolic strain



End-systolic strain corrected for size



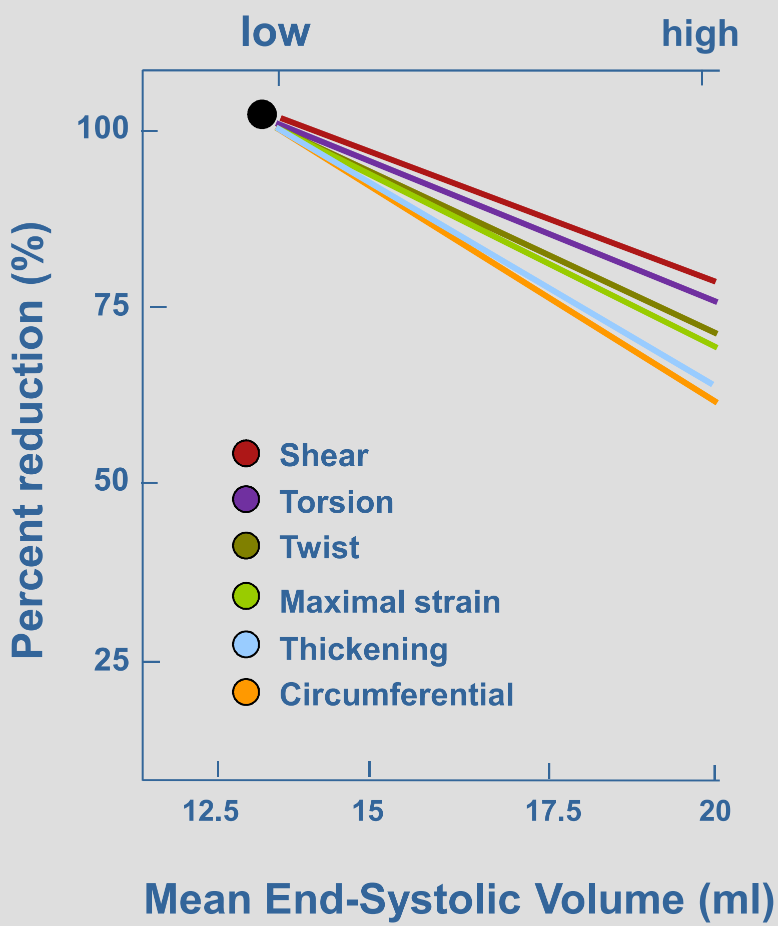
MV replacement criteria

Marciniak A, Claus P,... Eur Heart J. 2008

Deformation in increased afterload

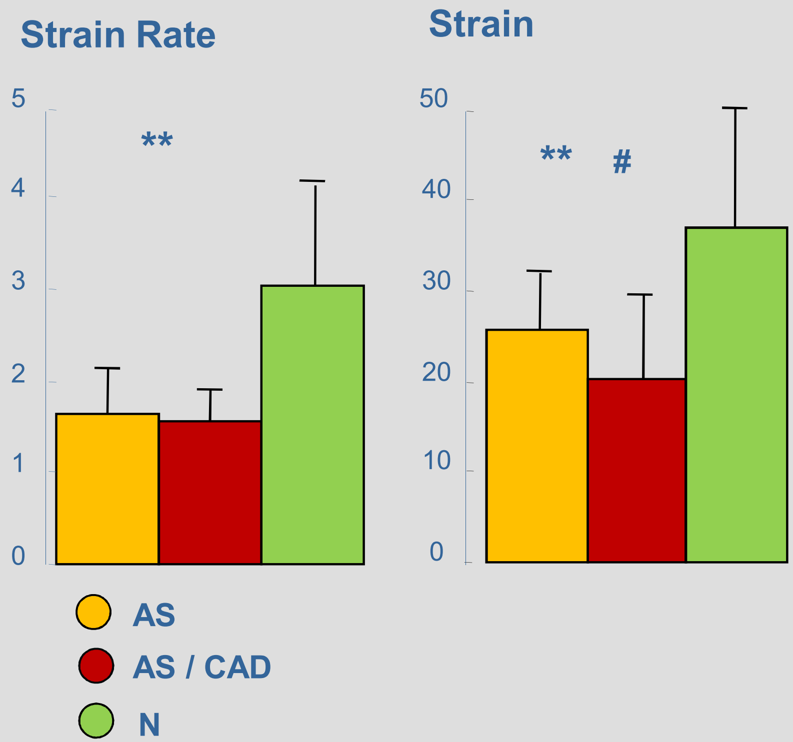
Experimental MRI

acuut



Clinical SR imaging

chronic

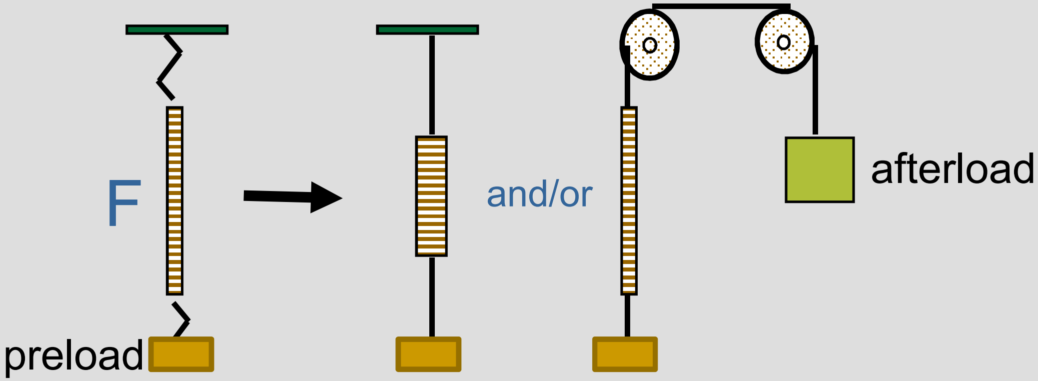


MacGowan 1996

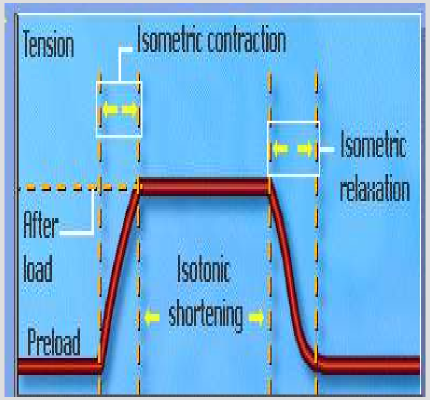
Kowalski, 2002

Myocardial Deformation and Loading

Defined in muscle strips...

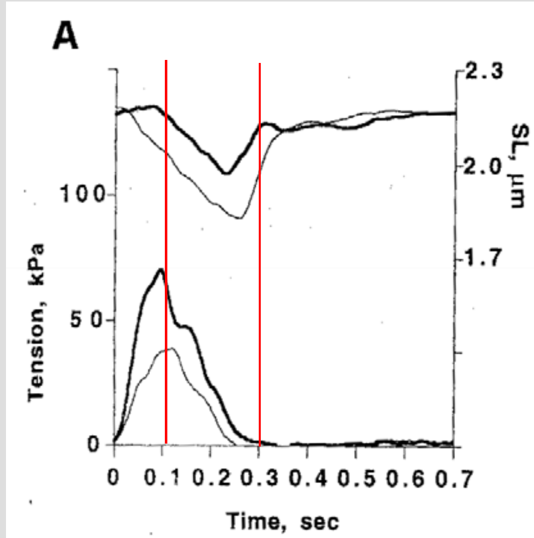


Shortening Force



In-vivo

Timing!

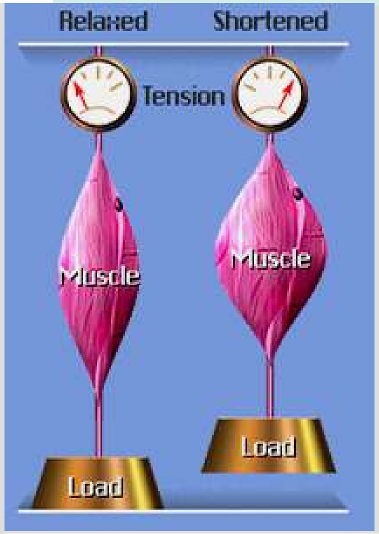


Guccione et al 1997

isometric - isotonic

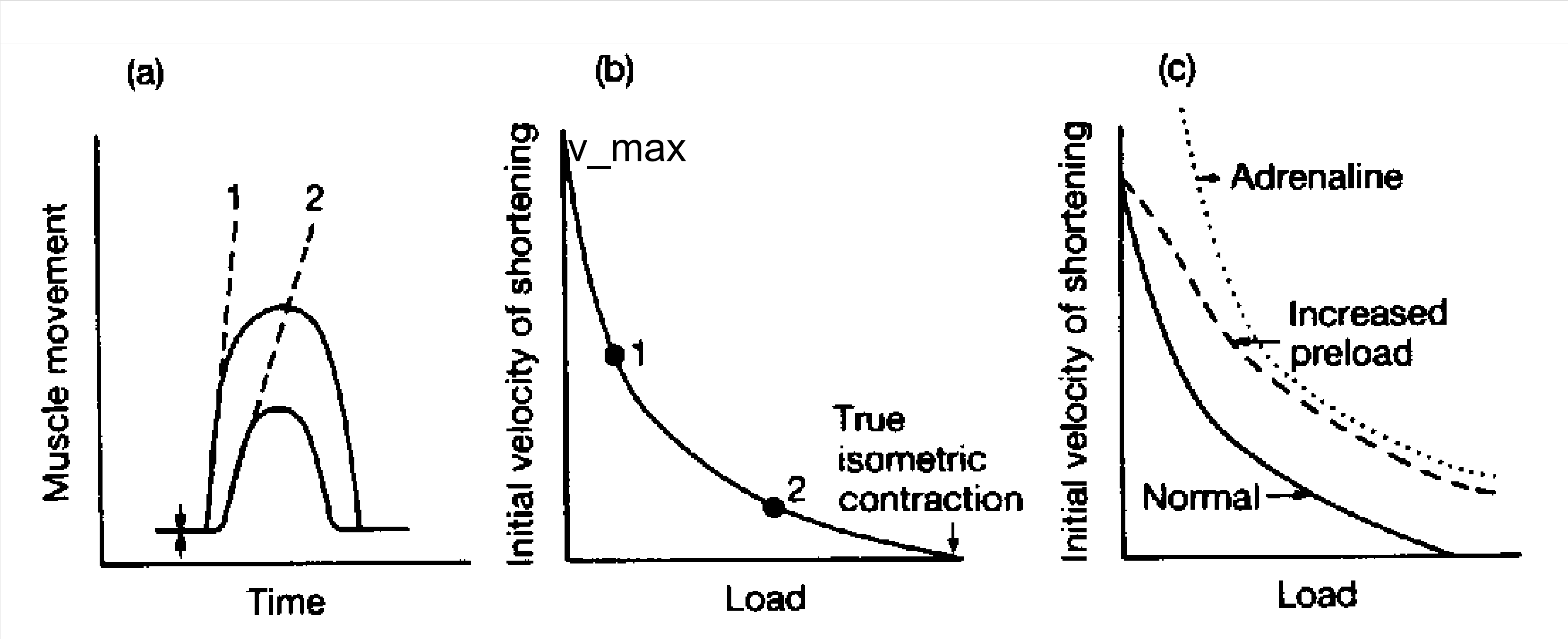
auxotonic

Velocity of fiber shortening



After-loaded contraction

v_max is a measure of contractility (potential to shorten at zero load).



Strain Rate

Fibres in the heart ...

Anderson & Becker: Cardiac Anatomy ‘80

- Laminar sheets
- Packed from base to apex
- Branching
- 4 cells thick
- Cleavage planes
- Tight coupling inside sheets

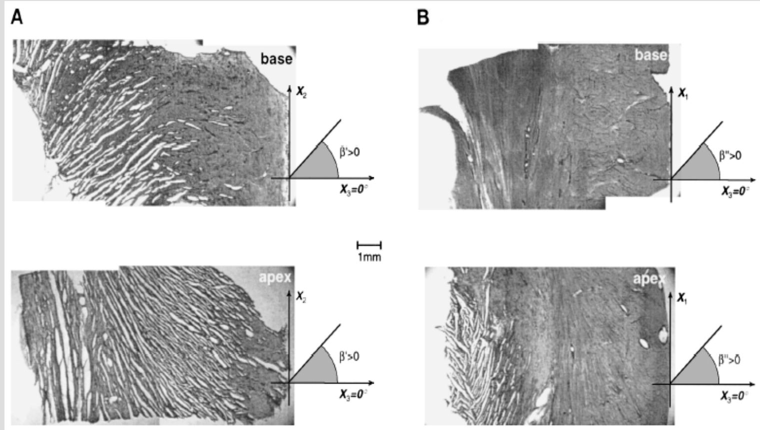
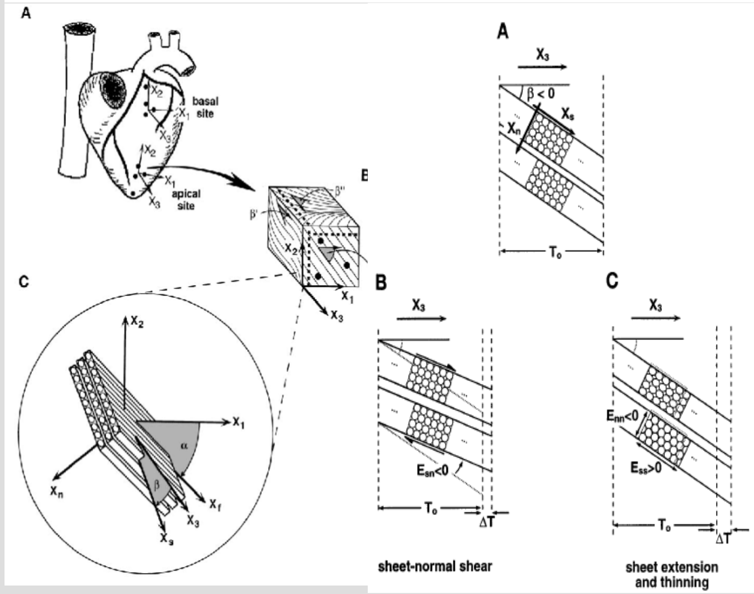


Fig. 2. Images of laminar cleavage planes in longitudinal-radial (*A*) and circumferential-radial (*B*) tissue sections from basal and apical measurement sites in anterior LV free wall. Each image is a montage of 2 video micrographs acquired under ×30 magnification. Orientation of specimen is indicated by cardiac coordinate axes X_1, X_2, and X_3, with endocardium to *left* and epicardium to *right*. *Study AF3*; LV pressure during fixation, 8 mmHg; section thickness, 50 μm; scale bar, 1 mm.



Mechanism for thickening

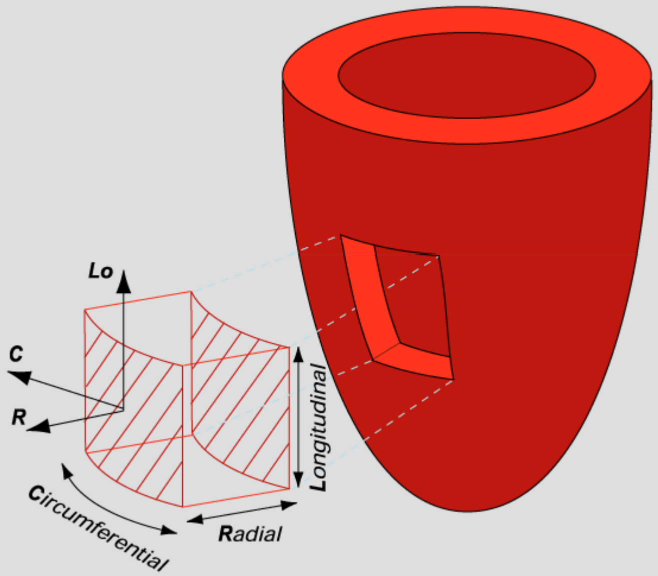
LeGrice 2001

Put all this in the wall of a heart

We have to describe this in the complex 3D setting of the heart.
But basic principles can be translated.

Cardiac system

- Radial
- Longitudinal
- Circumferential

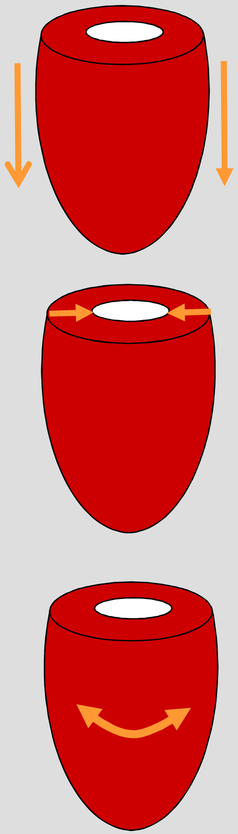


Intrinsic / Physiological system

- Fibre
- Fibre-Sheet
- Sheet-Normal

Required for correct physiologic interpretation !!!
(e.g. RR = wall thickening; CC/LL = circumferential/longitudinal shortening)

Deformation modes



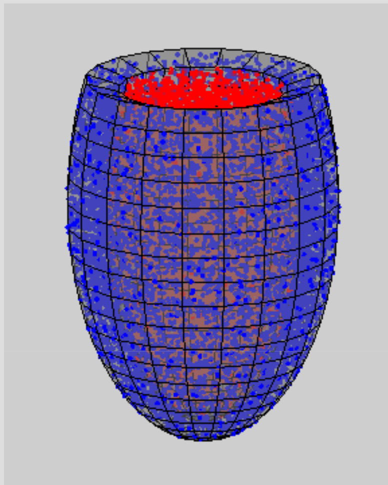
Longitudinal shortening

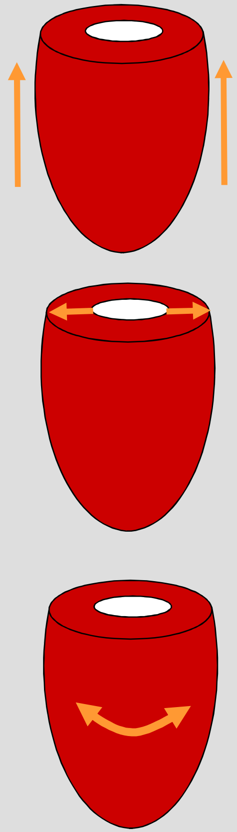
Radial thickening

Circumferential shortening

+

Endocardial inward motion for ejection





Longitudinal lengthening

Radial thinning

Circumferential lengthening

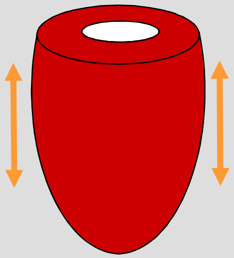
+

Endocardial outward motion for filling

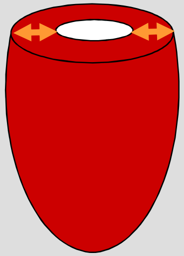
+ Shearing (twisting,…) optimize transmission of fiber force/deformation

Explain changes in deformation modes

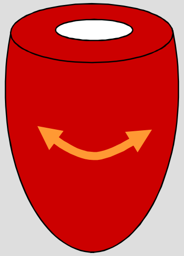
What we observe...



Longitudinal shortening

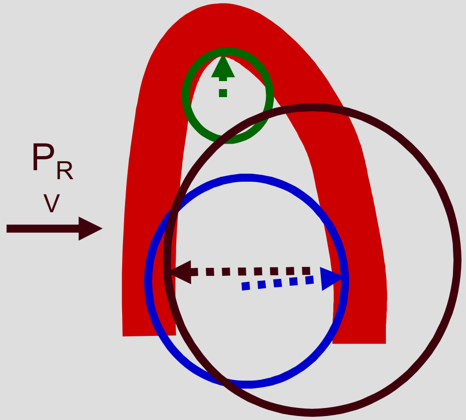


Radial thickening



Circumferential shortening + torsion

Why we observe this



LV wall stress:

σ= P x R / 2 h

Long axis shortening is differently affected by increasing load.

M mode EF remains normal or is even supranormal and does not pick up longitudinal motion

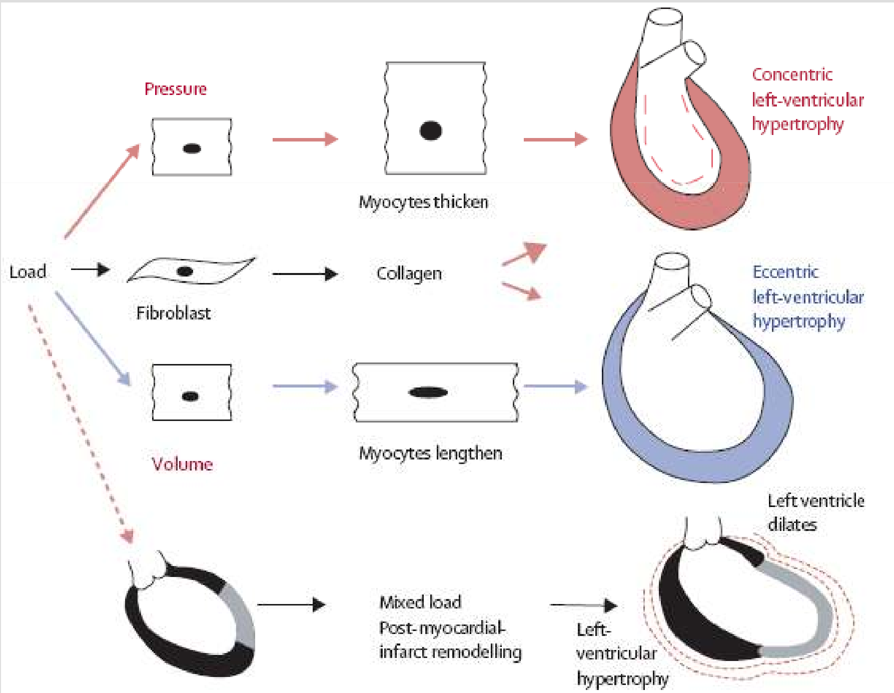
Remodeling

Ventricular remodeling

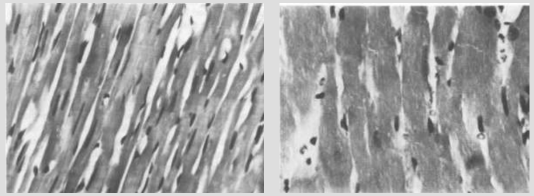
Cardiac Pathologies: ventricular remodeling → heart failure

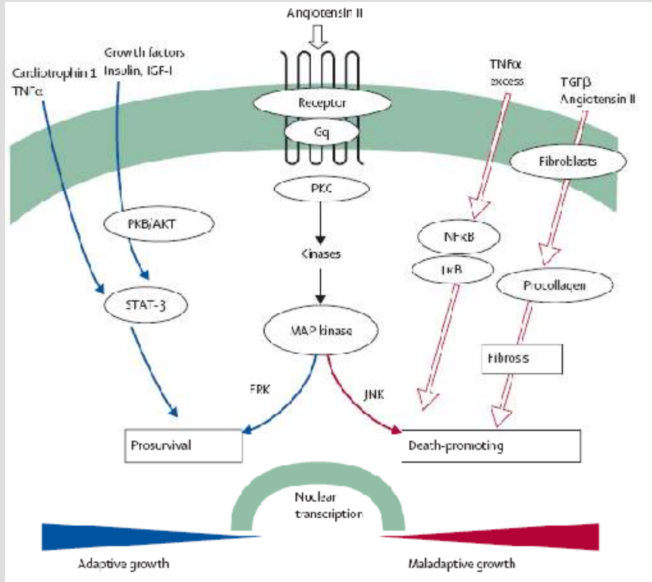
Chronic change in (regional) loading → structural changes [trigger]

Different patterns observed:



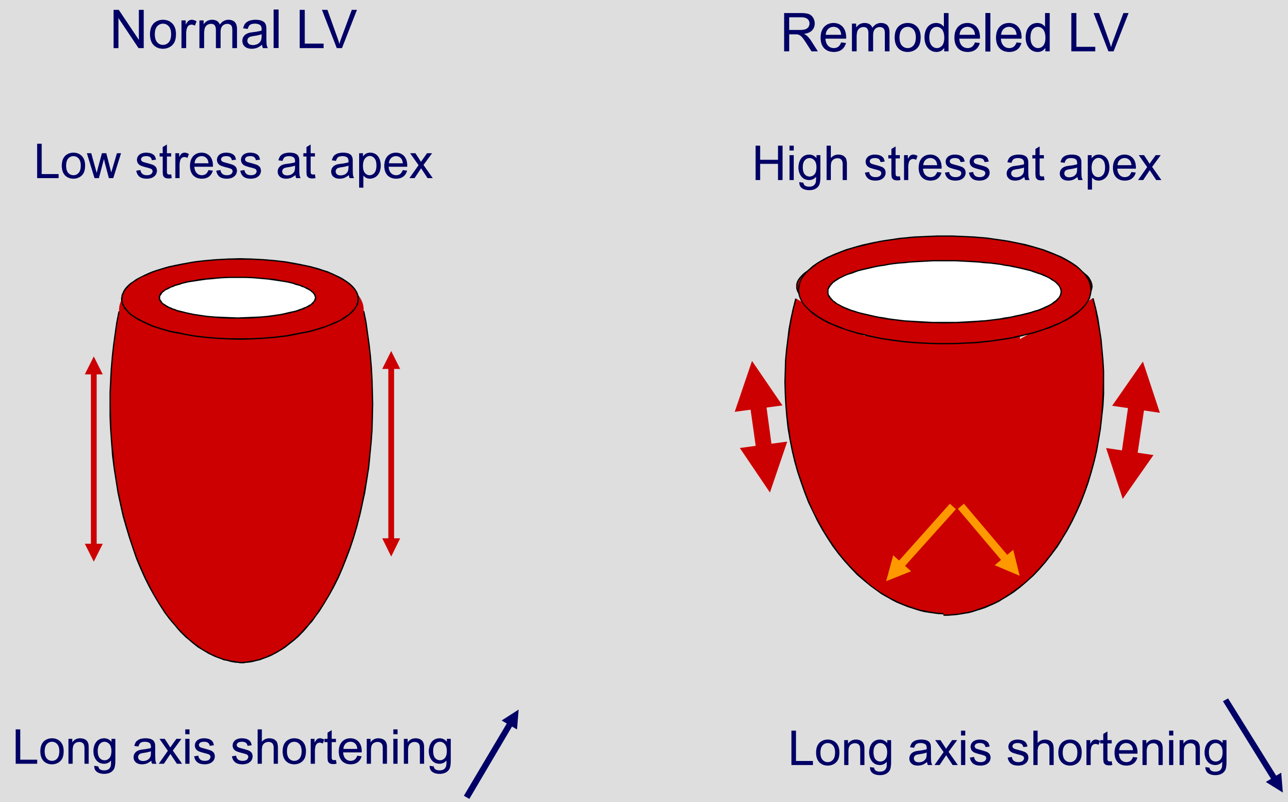
Opie et al. 2006





Influence on function: myocardial deformation?

Understanding deformation in pressure overload

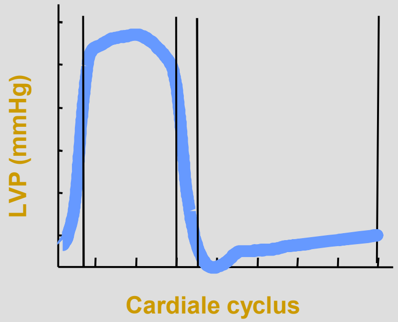


Always interpret deformation in relation to loading.
'Qualitatively' correct for loading. Quantitative correction not yet established.

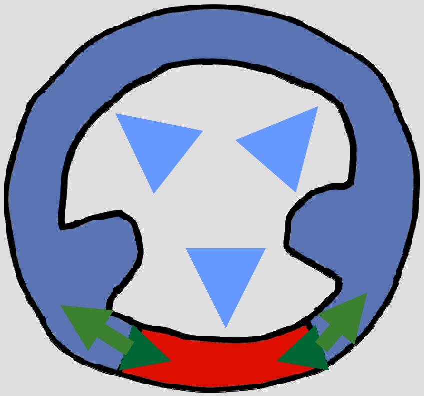
Active tension

Force balance in a myocardial segment

Total wall stress



pressure+geometry

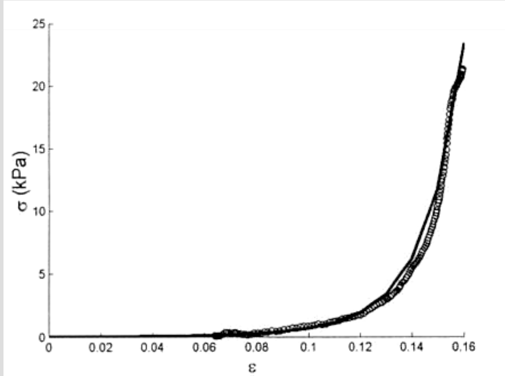


FP + FA = FE

Elastic force

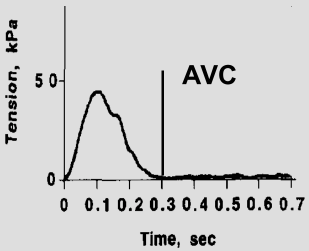
non-linear stress/strain relation $\sigma_E = C_1\,\varepsilon_c \exp(C_2\,\varepsilon_c^2)$

Guccione et al. J. Biomech. 28(1) 1994



Active force development

Expected behaviour



Guccione et al. J. Biomech. 30(2) 1997

Material constants ?

During diastole → FP = FE :

Fit exponential function to stress/strain data → C_1 en C_2

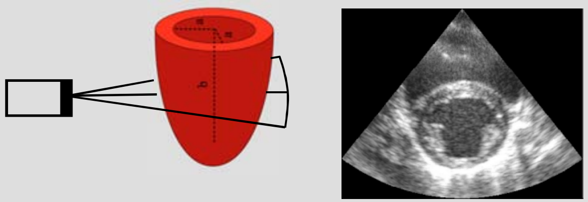
Active Force ?

During systole → FP and FE are known:

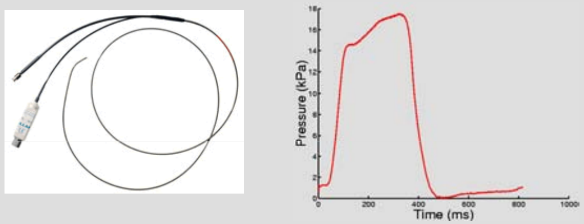
FA follows from force balance.

Normal active tension profiles

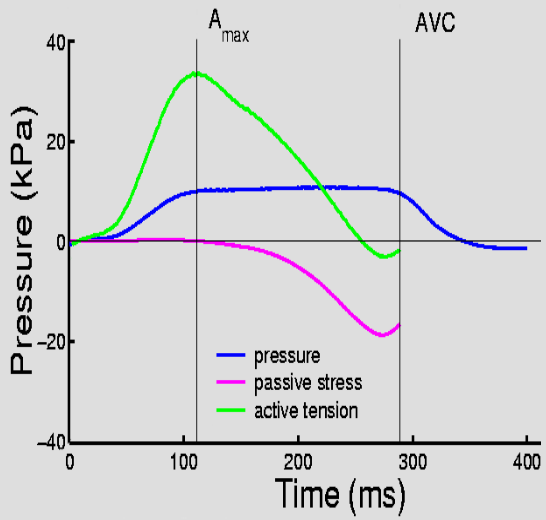
Deformation Imaging



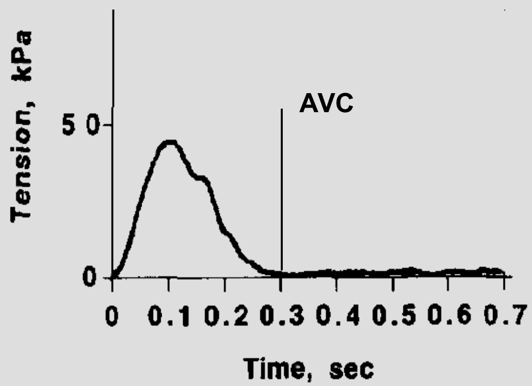
Pressure Measurements



Parameter	Mean ± SD
A_{max}	46 ± 9 kPa
T_A_{max}	113 ± 14 ms
AVC	303 ± 32 ms
C_1	2.52 ± 5.03 kPa
C_2	286 ± 125



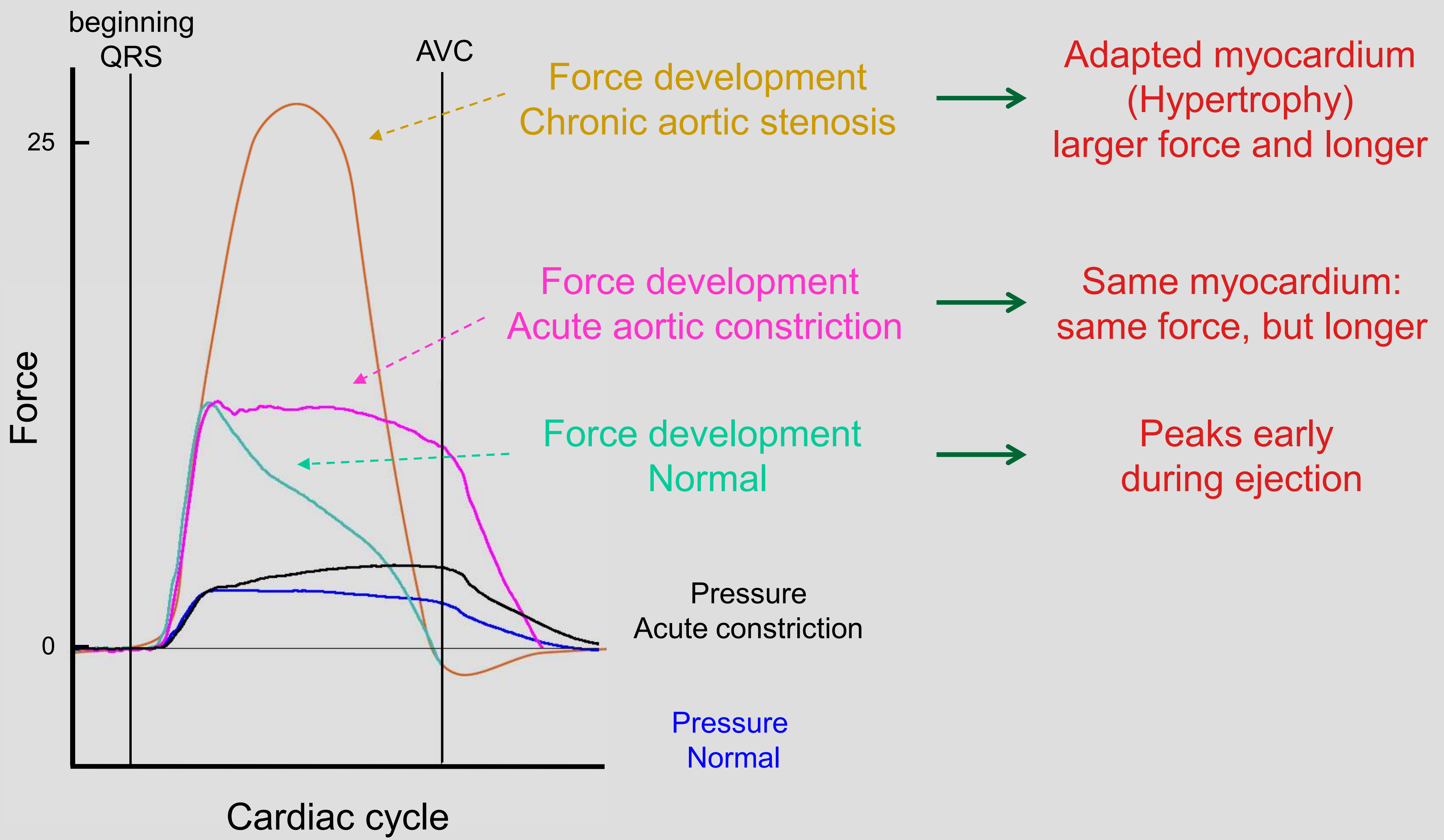
Forces measured in normal porcine model



Isolated muscle experiments
physiological loading conditions

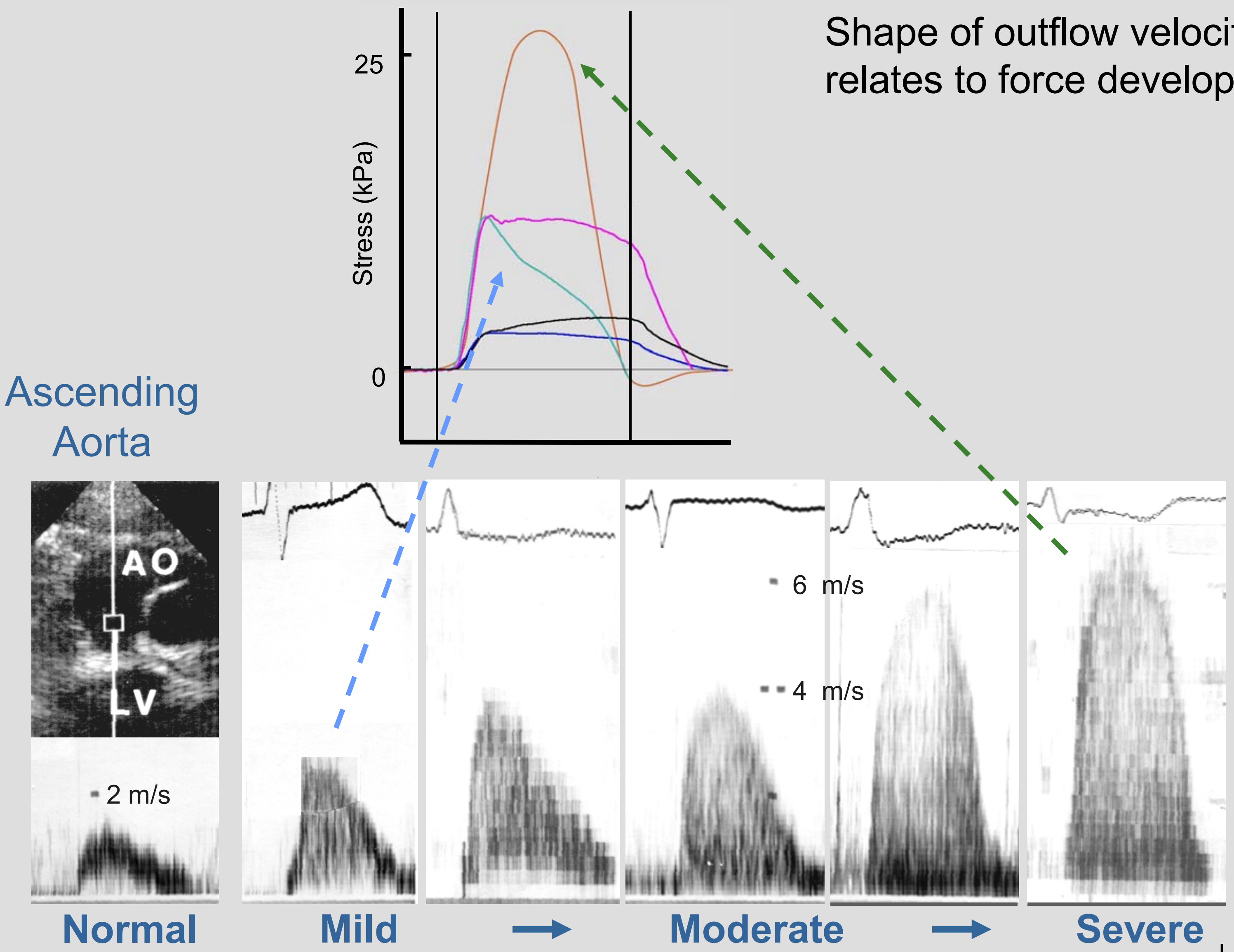
Adaptation to pressure overload: acute ↔ chronic

Both acute (beat to beat) and chronic pressure overload (Aortic Stenosis) reduce deformation, but what with force development



Force development translates in outflow

Shape of outflow velocities relates to force development



Preload and shape

Relation between *ventricular shape* and *passive mechanical properties*

Assumptions

1. Passive filling phase determines end-diastolic wall-stress.
2. Diastasis is the stress-free state (stress and strain = 0).
3. Myocardium is anisotropic with strain-energy function:

$$\Phi = (C/2)\,(\exp(Q) - 1)$$

$$Q = b_1 E^2_{FF} + b_2(E^2_{CC} + E^2_{RR} + E^2_{CR} + E^2_{RC}) + b_3(E^2_{RF} + E^2_{FR} + E^2_{FC} + E^2_{CF})$$

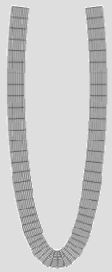
Guccione et al. (1991) Journal of Biomechanical Engineering 113(1), 42-55.

4. Transmural distribution of fiber orientation is linear from 75° to -45°.

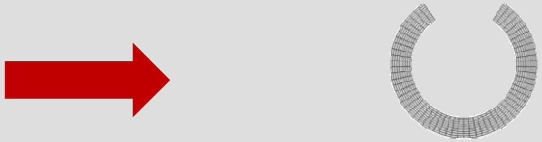
HF Choi, J. Biomechanics 2010

Simulations

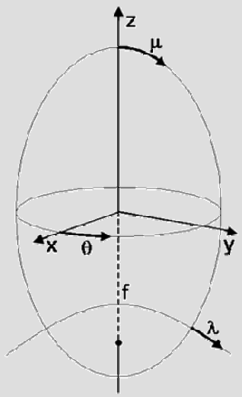
Shape changes:

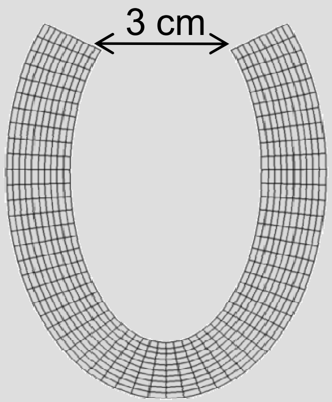


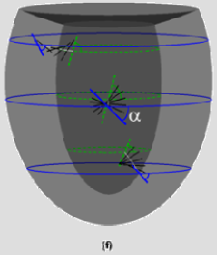
ELONG



SPHERE







Simulation details

Coordinates

- Prolate spherical coordinates

Elements

- 10 elements radial
- 30 longitudinal
- 1 circumferential

Boundary conditions

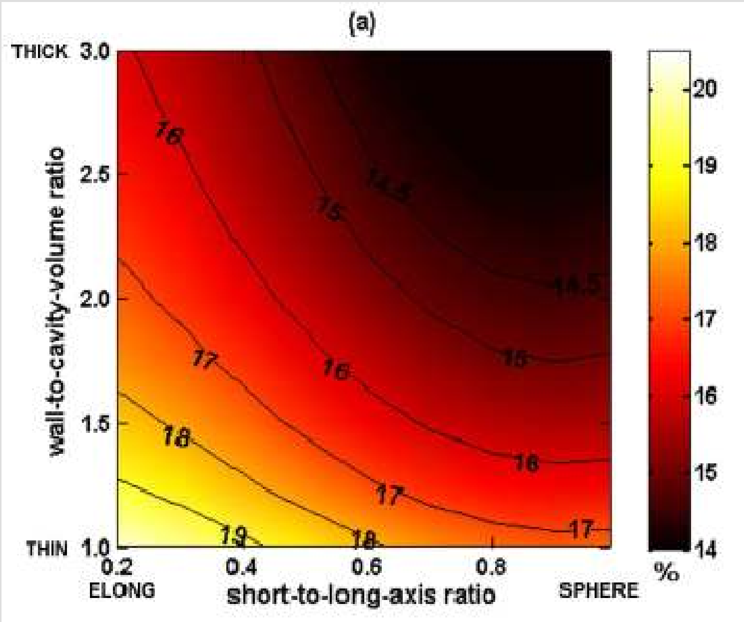
- base fixed
- conservation of myocardial volume
- 85 ml volume (rest: diastasis)
- Inflate to 8mmHg

Material

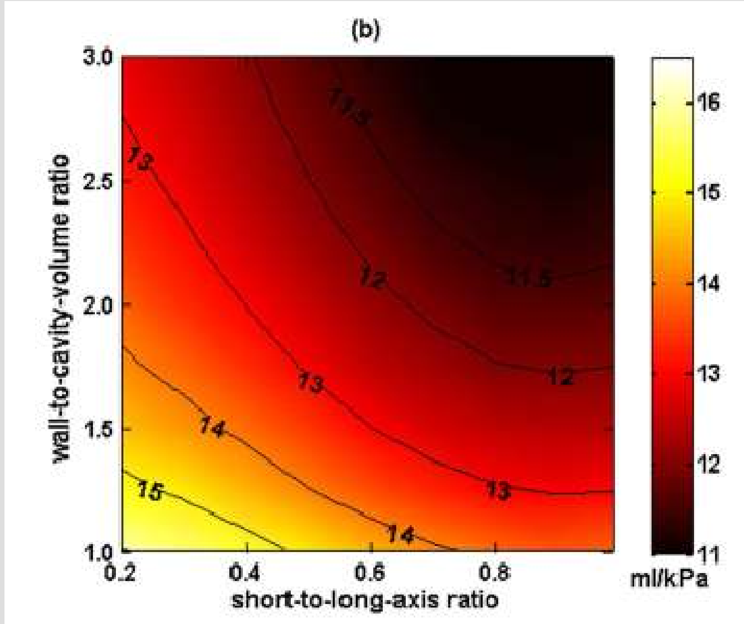
- Anisotropic
- Fiber orientations template

HF Choi, J. Biomechanics 2010

Global measures



Filling fraction



Compliance

Little changes in these global parameters, but...

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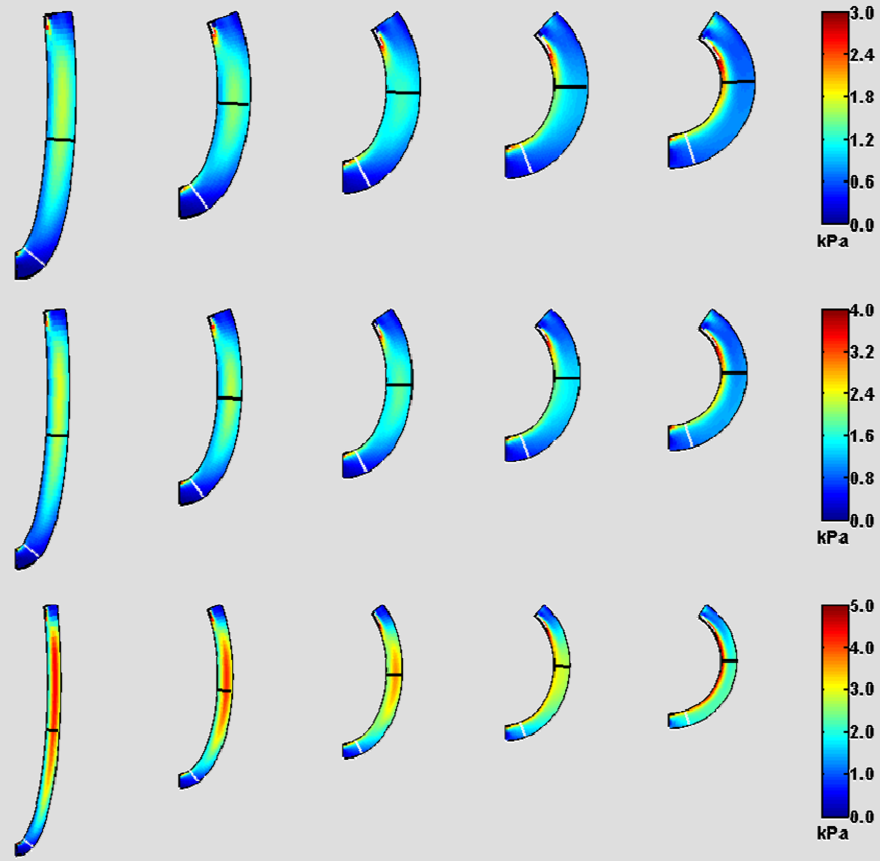
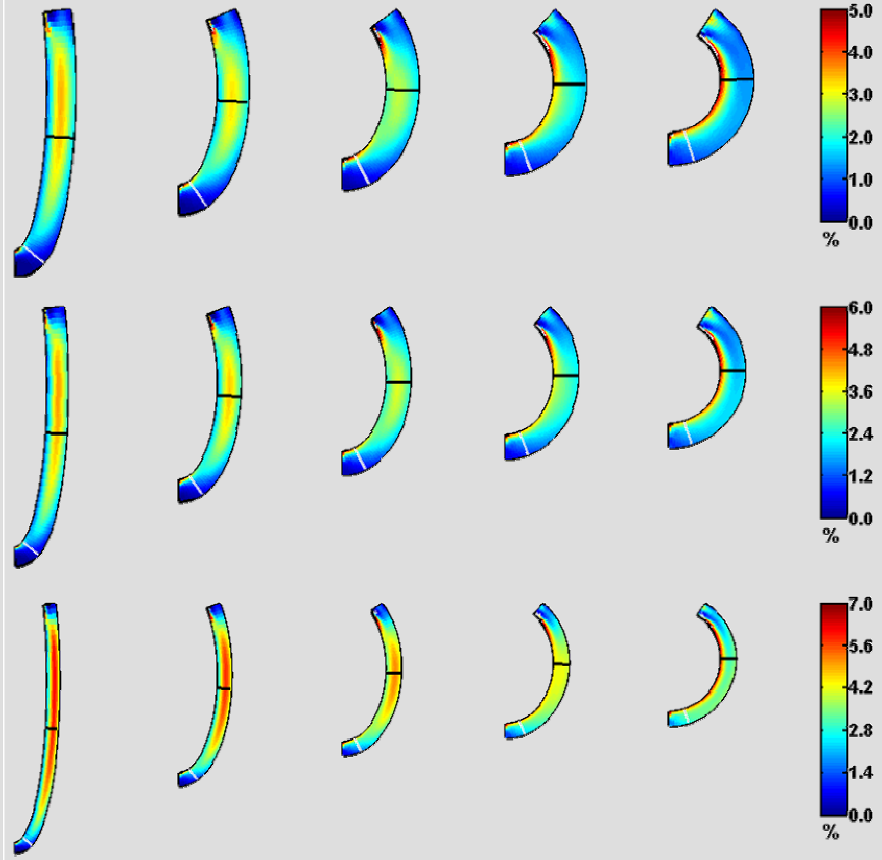
Fiber stress and strain



Fiber stress (kPa)

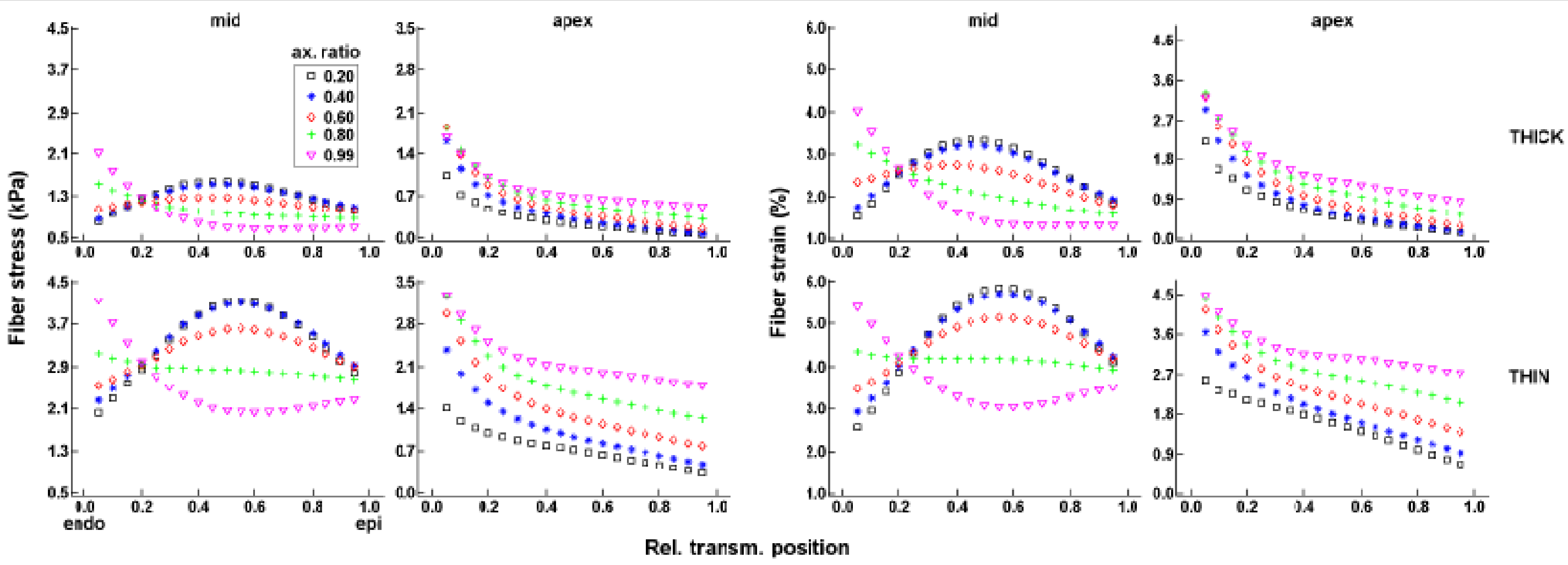


Fiber strain (%)

HF Choi, J. Biomechanics 2010

Fiber stress and strain

Influence of sphericity on transmural distribution in mid and apical segments



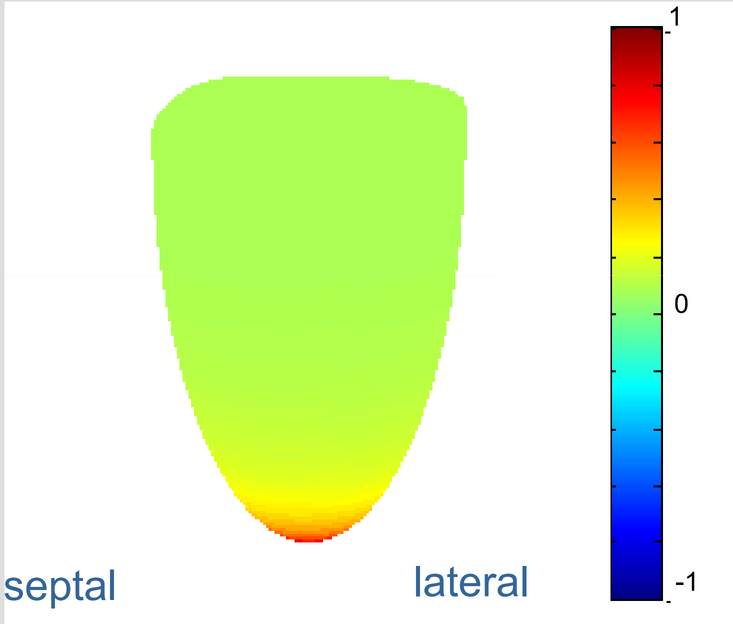
1. Fiber preload depends on shape.
2. Higher preload in regions with smaller curvature.

HF Choi, J. Biomechanics 2010

Geometry: LV is not an ellipsoid

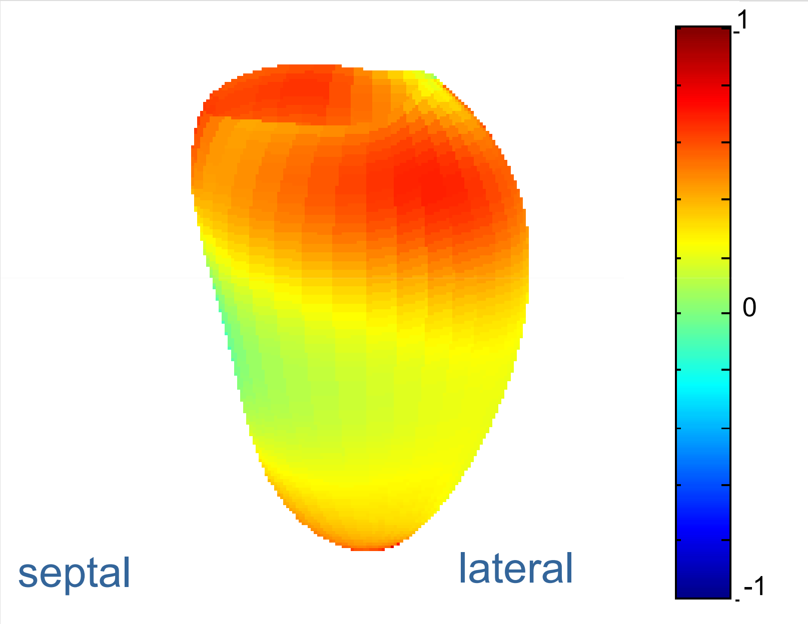
Normal left ventricular midmyocardial surface from a young healthy person (30y) based on 3D echocardiographic dataset.

NOT



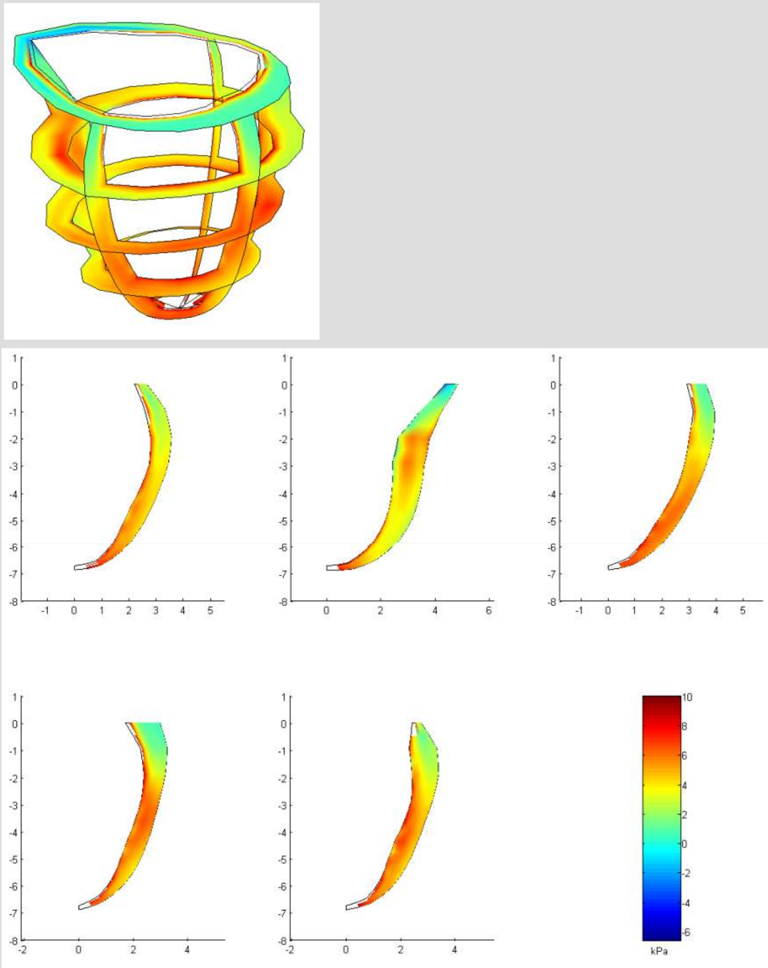
Closest fitting ellipsoid

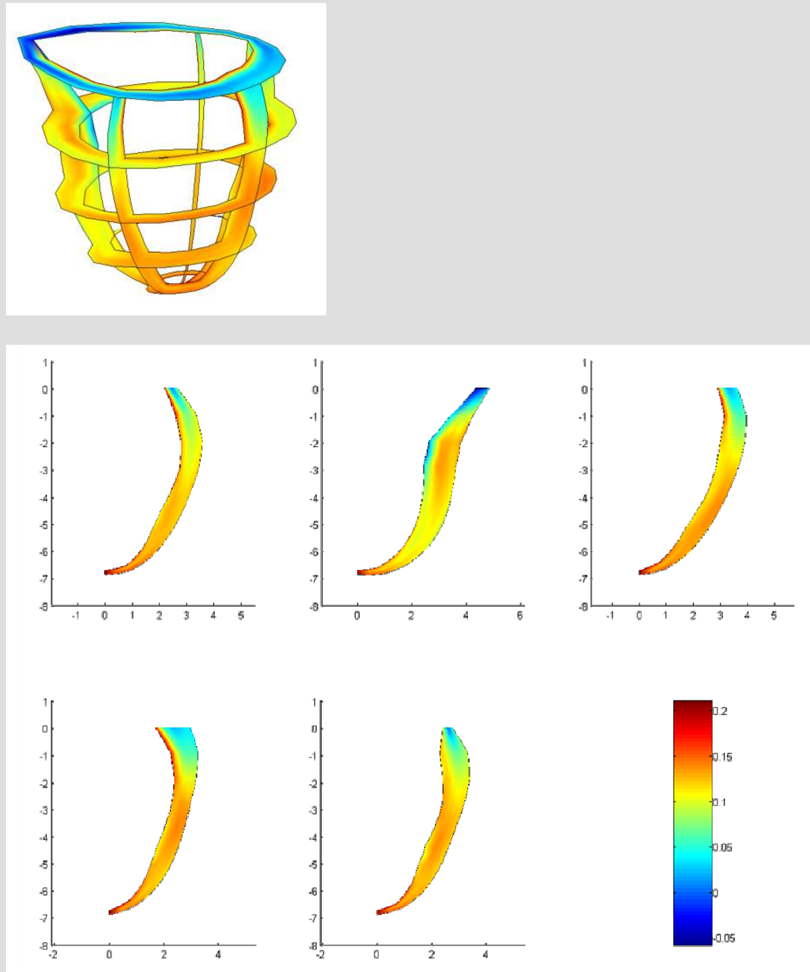
BUT



Curvature changes regionally.

Realistic geometry: fiber stress and strain

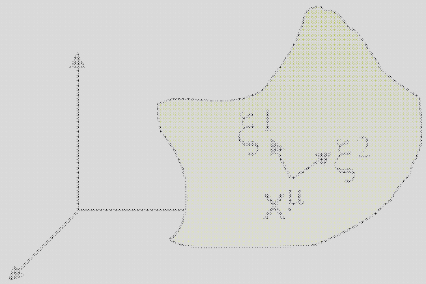




Realistic geometry causes more transmural homogeneity

Regional shape description: methodology

Embedding and induced geometry



Surface is locus of points parametrized by

$$x^\mu = \mathcal{F}^\mu(\xi^i), \quad i=1,2$$

Basis of tangent vectors $e_i^{\,a} = e_i^{\,\mu} E_\mu^{\,a} = \partial x^\mu/\partial \xi^i \, E_\mu^{\,a}$

3D 'analytic' representation of the surface

and define induced metric as (first fundamental form) $ds^2 = d\xi^i e_i^a \delta_{ab} e_j^b d\xi^j = d\xi^i g_{ij} d\xi^j$

and shape operator as (first fundamental form) $S_{ij} = (\partial^2 x^\mu/\partial \xi^i \partial \xi^j - \Gamma^\mu_{\nu\rho} e_i^\rho e_j^\nu) E_\mu^a N_a$

$$n^a = \tfrac{1}{2} \varepsilon^{ij} \varepsilon^a_{\ bc} e_i^b e_j^c, \qquad N^a = n^a/|n^a|$$

Curvature tensor of embedded surface $K^i_j(\xi) = g^{ij}(\xi) S_{ij}(\xi)$

Eigenvalues are minimal and maximal curvatureart the point defined by ξ^i

$\kappa_1 = 1/R_1$ $\mathcal{H} = \kappa_1 + \kappa_2$ (mean curvature)

Straightforward description of the curvatures

$\kappa_2 = 1/R_2$ $\mathcal{G} = \kappa_1 \kappa_2$ (gaussian curvature)

We define local sphericity index (more independent from size)

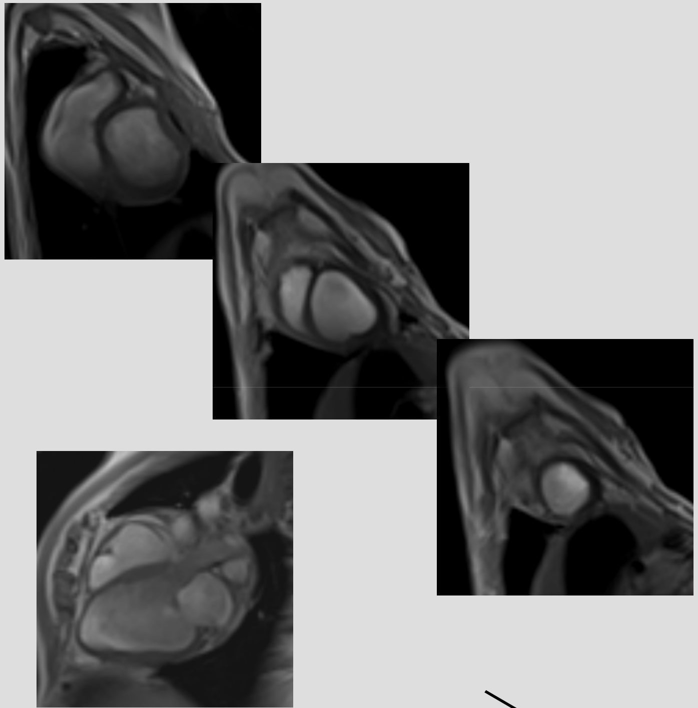
$$\mathcal{S} = \kappa_1/\kappa_2 = R_2/R_1$$

- Role for 3D echocardiography

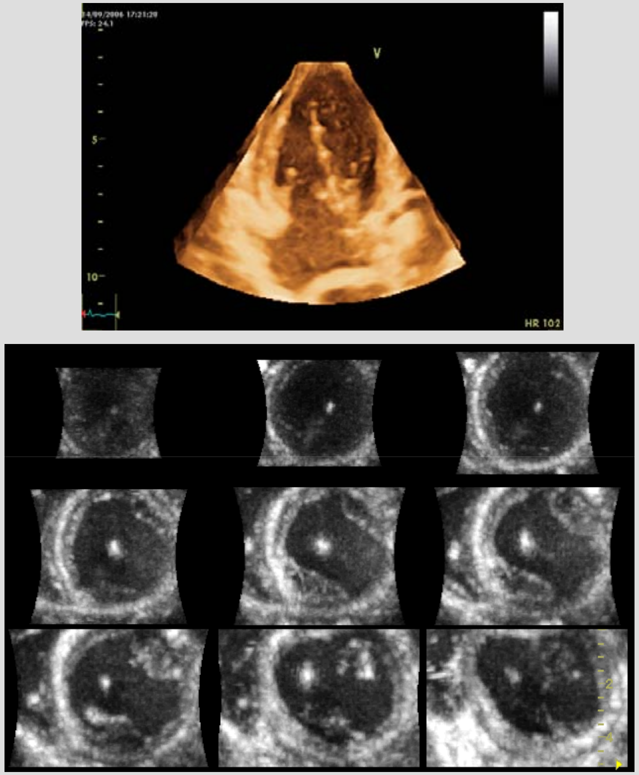
- Relevant and correct segmentation is important (manual vs automatic).

Imaging modalities

Magnetic Resonance Cine Imaging



3D echocardiography



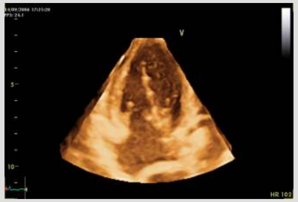
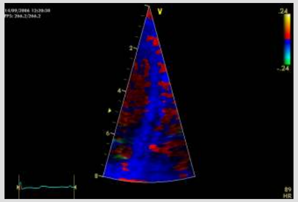
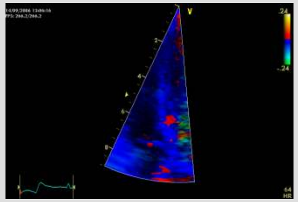
Segmentation/contouring

↓

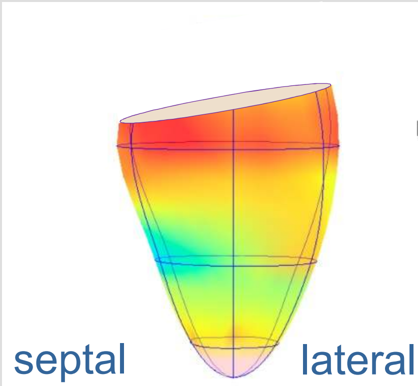
Point clouds (x^{μ}_{D}) of epicardial and endocardial surface

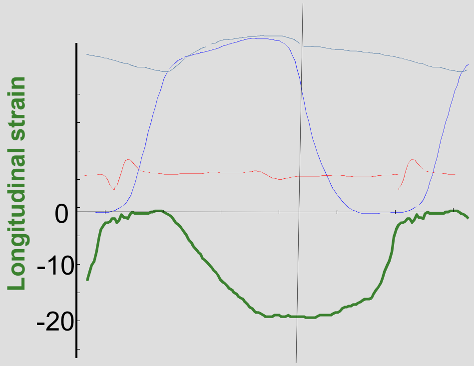
Regional shape versus regional function

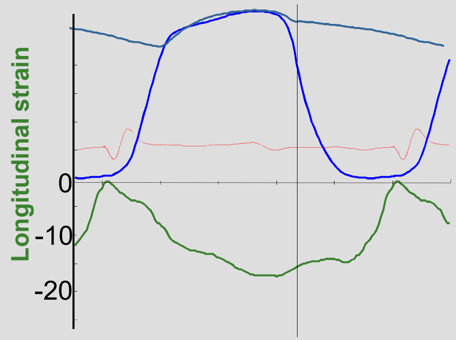
- → PRSW (9.3±1.8 to 8.9±2.0 mWatt.s/ml)
- → HR (106±10 to 108 ±11 bpm)
- ↘ CO (3.8±0.5 l/min to 3.3±0.4 l/min)
- ↗ EDV (90±15 to 103 ± 20 ml)
- ↘ SV (36±11 to 31±8 ml)

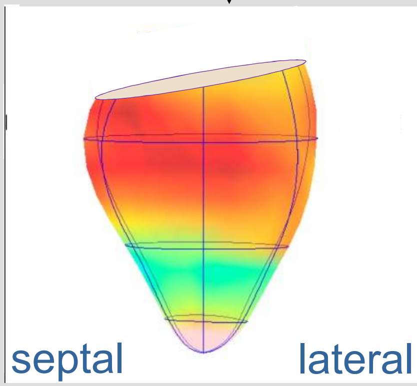
Baseline

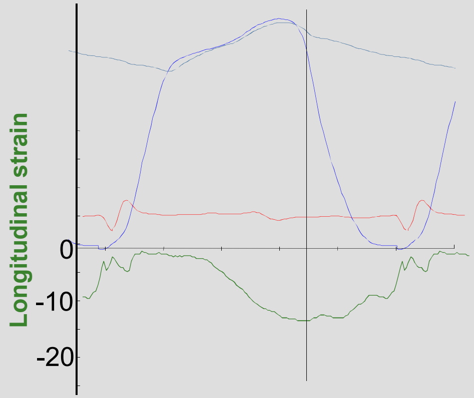


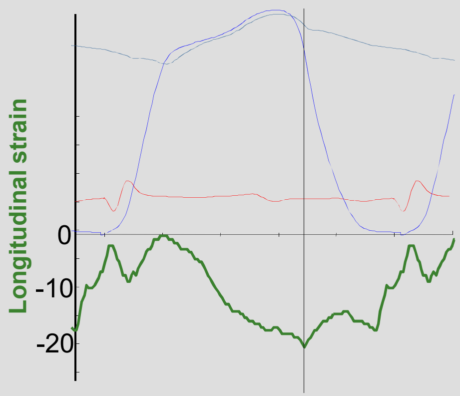




Acute aortic constriction







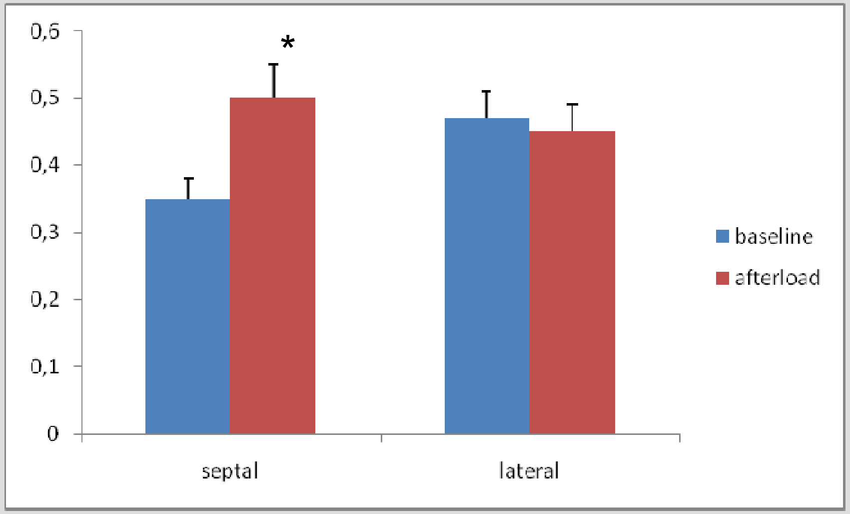
↘ SR (2.1± 0.4 to 1.3 ± 0.6)

Lateral **Septal**

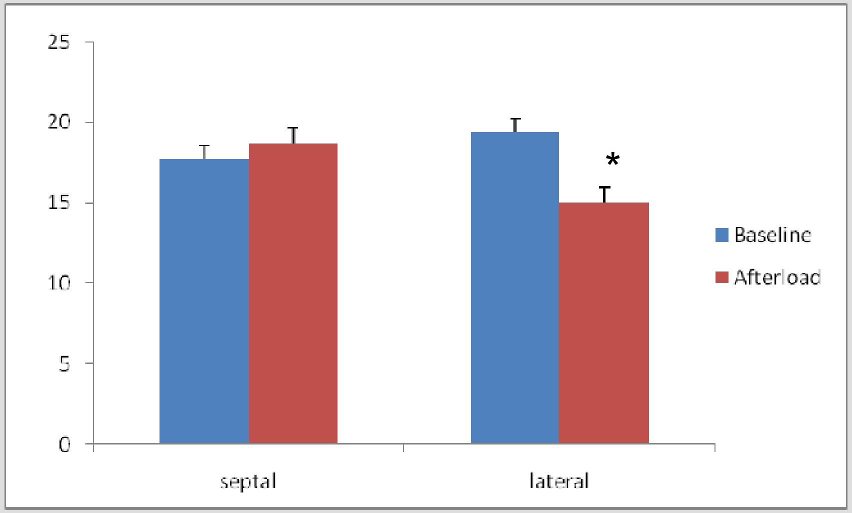
Regional shape versus regional function

Mid wall level

Regional sphericity



Longitudinal strain



In acute afterload change in regional shape is reciprocal to changes in deformation.

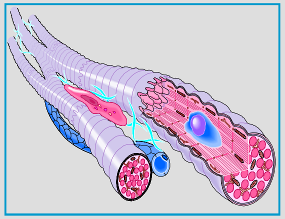
Regionally geometry can change to reduce decreases in function.
Spherical dilated hearts have no reserve ...

Conclusion

Perfusion
Activation

Systolic function: putting parameters in perspective…

active force development within the fibers depends on pre-stretch 1



2
cavity pressure development

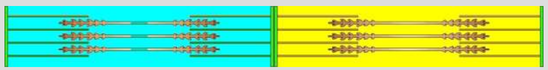
Regional mechanical boundary conditions

FIBRE ORIENTATION
Fibre rearrangements
for optimal pressure built-up
(shape change during IVC)

15% FIBRE SHORTENING
TO 60% EJECTION FRACTION

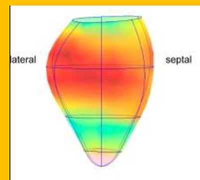
TISSUE CHARACTERISTICS
e.g. ELASTICITY
(collagen matrix, titin)

SEGMENT INTERACTION



GEOMETRY
shape, wall thickness

Volumes (preload)



Systemic
resistance
AFTERLOAD

3
Ejection by
wall deformation

Global mechanical boundary conditions

Abnormalities do not always affect ‘how much’ (symptoms) but will affect ‘how’...
remodeling